The Southern Surgeon

Subscription in the United States, $5.00

Vol. IX, No. 8

Copyright 1940 by
The Southern Surgeon Publishing Co.
August, 1940

INDICATIONS FOR SURGICAL TREATMENT OF INTRACRANIAL TUMOR

E. Bates Block Memorial Lecture

PERCIVAL BAILEY, M. D., PH. D.
Professor of Neurology and Neurosurgery
University of Illinois
Chicago

THE surgical treatment of intracranial tumors has developed remarkably and become generalized in the last thirty years. The methods of diagnosis, of localization, of operative technic have been fairly standardized. Most of the large American and European cities have now one or more neurosurgical centers where from 50 to 300 craniotomies are done yearly with partial or complete removals of intracranial neoplasms. Perhaps as many as 3,000 such operations are performed yearly throughout the world. But in spite of this imposing figure there are still physicians and even neurologists who are opposed to this branch of surgery. This antagonism is such, in the case of certain neurologists, that they advise against any intervention for intracranial tumor.

And, as a matter of fact, one can find a reason for this state of mind in the elevated mortality and the number of physical wrecks which emerge from certain clinics. One is reminded of the famous quip concerning the National Hospital for Paralyzed and Epileptics in London. Some wag, in the time of Victor Horsley, said it was so called because the patients entered epileptic and left paralyzed. The number of publications which permit us to judge adequately the results of surgical treatment for intracranial tumor is very small. The only adequate studies, apart from those of van Wagenen, Cairns, and Eisenhardt, from the clinic of Cushing, are the old ones of Tooth more than thirty years ago. In Cushing's clinic, according to Cairns, the operative mortality was around 14 per cent whereas ten years after operation 40 per cent of the patients were still living and 23 per cent enjoyed a useful life. If one compares such results with those of cancer in general they seem very favorable.

Delivered in Atlanta, March 18, 1940.

But one should have available similar data from other clinics. One of my pupils spent six months each in five different neurosurgical clinics in Europe and America and kept data concerning the mortality in each. The figures were as follows: 18 per cent, 30 per cent, 36 per cent, 44 per cent and 51 per cent. The number of survivors who were badly crippled varied also, but the short period of observation made it impossible to obtain valid data on end-results. Cairns believed that the difference in operative mortality could be explained by differences in manual dexterity and in the organization of the various clinics. Such an explanation is not valid for the above figures. The surgeons were all pupils of the same master and all of them of apparently equal manual ability. Their operating rooms were copies of the master's and they operated in much the same way.

This wide divergence in operative results arises from a difference in fundamental surgical philosophy. Two schools are sharply opposed. One, of which Dandy is the apostle, guides itself uniquely from the point of view of the treatment of cancer. Considering that a non-extirpated tumor is inevitably fatal it ignores the mortality-rate of operation and the resultant crippling of the patient. This attitude logically results in an attempt to extirpate practically all tumors regardless of their location, of their nature or of the condition of the patient. Thus one finds in the literature records of extirpations of entire cerebral hemispheres, even of the left hemisphere of a right-handed person. Such interventions, of course, result in a purely vegetative life for the survivor. The outcome of this attitude is a large number of operations, an elevated operative mortality and a large percentage of invalids among the survivors.

The other school attempts to estimate the operability of the patient at the time of entry to the clinic. In accordance with the volume and extension of the tumor, the functional importance of the region in which it is located, and the nature of the tumor itself, the surgeon intervenes radically, partially, palliatively, or abstains entirely. From such a point of view the number of operations will be less, but the operative mortality will be lower and the defects provoked by operation fewer. The great master of neurosurgery, Cushing, always had the old maxim *primum non nocere* in mind and struggled all his life to reduce the operative mortality under 10 per cent.

In the results of the two schools the number of definitive cures with useful survival may be about equal when corrected for the total number of patients having intracranial tumors whether operated or not. But we must ask ourselves whether the radical school might not, in spite of its elevated immediate mortality, from time to time

save a patient who would have been abandoned by the conservative school. If so it could be used as an argument for the radical attitude. On the contrary the elevated mortality of that school is harmful for the future of neurosurgery and tends to precipitate the decline of this specialty. It would seem therefore wise to attempt to determine more accurately the indications for operation so as to maintain as low an operative mortality as possible, at the same time making sure that no curable case is overlooked. It is an essay in this direction which I wish to present to you this evening.

The problem of indications for operation in the case of intracranial tumor has not received the attention it deserves. The solution of this problem can alone place cerebral surgery on the same plane as abdominal, thoracic or orthopedic surgery. It is evident that the indication to operate must be based here, as elsewhere, on the physiology of the organ on which one intervenes and on the nature of the lesion attacked. We will discuss first the physiologic considerations.

Interventions have been made on practically all regions of the brain. Only the bulb and pons have been rather consistently avoided, but elsewhere the surgeon has not stayed his hand, even going so far as to remove almost an entire cerebral hemisphere, at least the cortical portions of it. But it is obvious that the thesis that every brain tumor should be removed whatever its location and whatever its nature cannot be pushed to its logical extremity any more than one can amputate through the first lumbar vertebra for a gangrene of the lower limb which has extended to the abdomen. We must review briefly the various regions of the brain and try to determine what would be the results of surgical trauma to these parts.

The bulb and pons are so sensitive to surgical traumatism that they must be handled with extreme care for fear of respiratory paralysis or other complications. Tumors within them are inoperable. In childhood unfortunately gliomas develop frequently (15 per cent of all cases) within the pons. No attempt to remove such a tumor is ever justifiable. The establishment of the diagnosis is a definite contraindication to operation. Having several times made this diagnosis and allowed the patient to die without operation, I have never had occasion to regret my decision. Tumors in the neighborhood compressing the pons and bulb must be dissected away with great care. This is possible with the acoustic neurinoma, if one has a good exposure, but the dissection of an ependymoma in the fourth ventricle has in our experience always resulted in a disaster. Whenever such a tumor is identified now I content myself with a decompression.

The mesencephalon and hypothalamus are also very fragile and trauma to them is very likely to result in unconsciousness and hyperthermia. Any tumor invading or developing within these parts is not removable. But tumors may develop in nearby structures and be sufficiently encapsulated to be dissected away without too much damage. Such tumors develop from the paraphysis and pineal bodies and have in a few cases been successfully removed. Adenomas of the hypophysis may also be removed, since they are soft and fragile and have a smooth capsule, but craniopharyngiomas are more difficult because fingers of epithelial cells often extend into the hypothalamic tissue. In order to reach these regions one must plan the approach so as to do the least damage. The pineal region may be approached between the cerebral hemispheres, and through the splenium of the corpus callosum, section of which causes no apparent defect. In certain cases in which there is a large internal hydrocephalus an incision may be made through the right parietal region into the lateral ventricle and a pineal tumor removed by this route; one would not make such an approach on the left side for fear of an aphasia. Such an approach on the right side might result in a hemihypesthesia on the opposite side of the body, or a defect in the visual fields, but such a defect is not very disabling and can be justifiably risked. In certain cases an entire occipital lobe has been removed to give access to a pineal tumor; this results, of course, in a complete homonymous hemianopia.

The approach to a tumor in the hypothalamic region is also difficult. If there is a large hydrocephalus an opening may be made through the right frontal lobe and the tumor attained by enlarging one foramen of Monro. Such an approach on the left side might cause a motor aphasia; on the right side only a transitory facial weakness and inability to turn the eyes conjugately to the opposite side are to be feared. The hypothalamic region may be approached underneath the frontal lobe from in front, but this approach is so cramped for room that some surgeons deliberately sacrifice one optic nerve or split the chiasm in order to gain room to manipulate. The resultant defects will be obvious to you; the surgeon must judge whether the provocation of such defects is justified by what he can accomplish by causing them.

Whatever approach is used to attain the hypothalamic and mesencephalic regions the nervous tissues must be carefully protected. Failure to do this will result first of all in unconsciousness. It takes a very small lesion in these regions to arrest consciousness. The usual result is a very strange clinical picture. The patient after recovery from the anesthetic remains in a state which I can only describe as one of arrested consciousness. He seems to be quietly

resting and one has the impression that it is necessary only to shake him to make him arouse and talk. But on the contrary no amount of stimulation will obtain any response. There may be a grimace of the face when the patient is pricked with a pin, or even a whining cry, but nothing more. At the same time the temperature-regulating mechanism is upset so that the hyperthermia may demand active treatment. In some cases tonic cervical reflexes of Magnus-de Klejn may be obtained. After a few days the hyperthermia may subside and I have known patients to live in such a state for eighteen months. Of course, they had to be fed by nasal tube. When the hypothalamus is injured farther forward near the optic chiasm a diabetes insipidus may result. Because of these serious complications, which may accompany operative trauma to the hypothalamus and midbrain, operations in these regions must be conducted with great circumspection.

The cerebellum is more tolerant. The ablation of a large part of one hemisphere does not result in any important permanent deficit if the dentate nucleus be not injured. During the first days following such an ablation there is a marked asynergy of the homolateral limbs; this rapidly improves for a few weeks and then more slowly until after six months very little defect remains. Such an extirpation must sometimes be made in order to gain access to an acoustic neurinoma. The vermis of the cerebellum is more sensitive. A serious injury to the vermis results in marked disturbance of equilibrium so that walking may be impossible. Nevertheless the posterior part of the vermis may be split sagittally in order to gain access to the fourth ventricle without any permanent deficit remaining.

I shall not attempt to make a survey of the physiology of the cerebral hemispheres. I should like to point out that here as elsewhere in the central nervous system not only the region involved is important but also the mass of tissue removed or destroyed. Lashley demonstrated that the diminution of the faculties of a rat is in direct proportion to the quantity of cortical substance removed whatever the localization. This is not true of higher mammals and yet the quantity of tissue removed is important. Kennard and Ectors have shown that in the monkey only extirpation of area 8 (the prerolandic oculomotor region) causes a forced rotation of the eyes in the monkey, but the more cerebral tissue was removed the more evident became the symptoms, to become maximum in the hemidecorticated monkey. One must be economical of the cerebral tissue therefore, even in the so-called silent areas. Cortical ablations are now made not only to gain access to tumors but in the treatment of focal epilepsy and of athetosis, even of certain types of mental disturbance. But in all these cases it is important that the surgeon have a definite and accurate knowledge of the reasons for such extir-

pation and of its results, and be content with the minimum necessary to achieve his purpose.

There has been much discussion of the results of ablation of important masses of cerebral tissue such as the frontal lobes, occipital lobes or the right temporal lobe of left-handed persons. Jefferson maintains that no symptoms result from removal of the prefrontal areas. This has not been my experience. Halsted has proved beyond question by psychologic examination of my cases that even extirpation of one frontal lobe leaves defects of judgment which are easily demonstrated. Resection of the temporal or occipital lobes results always in a hemianopia but the other resultant defects are largely unknown and do not trouble the patient much when the extirpation has been on the right side. Of course, in right-handed patients aphasic difficulties are caused by such extirpations on the left side. Extirpations in the central or parietal regions cause paralysis and anesthesia depending on the location and extent of the lesion. One must be particularly economical of tissue in the precentral and postcentral gyri and around the Sylvian fissure on the left side.

So far we have talked only of actual extirpations of tissue, but functional extirpations may be made by ligature of arteries or important veins. The symptoms caused by occlusion of the various cerebral arteries have been studied for many decades and, while I do not believe with Dandy that occlusion of the left anterior cerebral artery causes permanent loss of consciousness, the occlusion of any major cerebral artery is serious and there is no excuse for a surgeon who ties one of them unnecessarily. The central group of superior cerebral veins must also be avoided lest a heterolateral crural monoplegia result and Cairns has found a hemianopia to follow occlusion of the internal occipital vein. The great cerebral vein of Galen, and its tributaries, the internal cerebral veins, must not be injured or occluded lest severe disturbances of consciousness and temperature-regulation result. In the approach to an acoustic neurinoma practically the entire venous drainage of the brain may be impeded by tying the lateral sinus, in case the opposite one is inadequate, with very serious results.

In addition to the effects of vascular occlusion it is wise to remember that prolonged compression of parts of the brain by retraction may cause great destruction at a distance so that in many cases it is better to gain access by a clean extirpation than by forcible retraction. Finally we should protest against the use of the electrical scalpel in the brain itself. It is unnecessary, since the brain-tissue can be cut readily by any blunt spatula. The electrical scalpel does not cut well in the subcortical tissue because of the high lipiodol con-

tent and, when used to stop bleeding, causes destruction along the vessels several centimeters in depth with resultant thrombosis and secondary softening. It is, on the contrary, indispensable for the intracapsular enucleation of meningiomas over the base of the cranial cavity.

For the above reasons it is necessary that the surgeon who operates within the cranial cavity be thoroughly familiar with the anatomy and physiology of the brain. Only a thorough training in neurology can give him this familiarity. For any surgeon who is interested in the condition of his patient after he has completed the operation this knowledge is important but not so much for the surgeon who looks on every intracranial tumor as a cancer to be extirpated at whatever cost. To decide whether the latter attitude is justifiable we must turn for a while to study the pathology of intracranial tumors.

Since the days of Rokitansky, Cruveilhier and Virchow the clinic has been based on pathology and, despite the recent development of biochemistry and bacteriology, pathologic anatomy remains one of the indispensable bases of the medical clinic. Why should it not be important also for the treatment of intracranial tumors? Nevertheless, in most of the classical treatises treatment does not seem to be very closely related to pathology. As eminent a specialist as Dandy affirms that the pathology of intracranial tumors is of no importance to the clinic. Sachs also minimizes the importance of pathology and even Cushing insinuated that the study of pathology is a regrettable influence. Certainly one cannot deny the importance of the technic of surgery, but I must insist that in the treatment of intracranial tumors, as of every other malady, pathology must be taken into account.

What influence can a knowledge of the nature of a tumor have on the conduct of our treatment? For example, let us consider the neurinoma of the acoustic nerve. This pathologic diagnosis remained without much importance as long as Cushing explored systematically the subtentorial region through a bilateral cross-bow incision in every case of tumor in the posterior fossa regardless of its nature. But if one refuses to accept this standardized procedure it becomes necessary to establish before operation not only the side on which the tumor lies but also its nature. The old bilateral cross-bow incision of Cushing was never an ideal procedure for acoustic tumor, so that Cushing himself rarely accomplished a complete extirpation of such a neoplasm, but contented himself with a partial intracapsular enucleation. In this way the immediate mortality was reduced but the symptoms recurred within a short period of months or years. For this reason new operative procedures have been imag-

ined by Dandy, Naffziger, Olivecrona and Bailey which permit complete removals. All of these newer procedures presuppose an accurate preoperative pathologic diagnosis.

In the case of tumor of the hypophysis Cushing himself based his operative procedure on the nature of the tumor. He operated on hypophysial adenomas by the transnasal route, at least until near the end of his career, and on craniopharyngiomas by the transfrontal route. At the present time the transnasal operation is almost universally abandoned, possibly because neurosurgeons are not familiar with the nasal cavities, and the problem has lost a great deal of its interest. Most adenomas can be successfully treated by roentgen radiation if diagnosed early and the late ones do not improve much anyway. The craniopharyngioma is very unfavorable for operative treatment; even the problem of saving eyesight is difficult in these cases. Frazier in his later years favored roentgen radiation of these growths, probably because of unfavorable surgical experiences; the experiences of others with roentgen radiation of craniopharyngiomas has not been encouraging.

Another tumor which occurs in the hypophysial region is the glioma of the optic chiasm. It may be difficult to differentiate in childhood from the craniopharyngioma. It is rarely calcified; but the craniopharyngioma may also not be calcified in about 15 per cent of the cases. The hypothalamic symptoms of the two tumors are superposable. It is important to recognize the glioma of the chiasm because it is almost always inoperable. Extirpation would result in blindness, and even simple exposure often results in a fatal hyperthermia. Even removal of the intraorbital extension of this tumor has caused death. Fortunately the tumor often causes enlargement of the optic foramina and is sometimes associated with the cutaneous manifestations of von Recklinghausen's disease. These features enable it to be recognized in many cases.

Finally small meningeal tumors grow from the tuberculum sellae. The syndrome which they cause has been described by Cushing as a progressive hemianopia (bitemporal) in a middle-aged person with normal sella turcica and no hypopituitary signs. They may be removed with greater or lesser difficulty depending mainly on their size.

So we see that, according to its pathologic nature, a tumor in the hypophysial region should be subjected to roentgen radiation in case of an adenoma, to surgical removal in case of a meningioma, to operation also perhaps in certain selected cases of craniopharyngioma and to complete abstention in cases of glioma of the optic chiasm.

During childhood subtentorial tumors are particularly frequent. These tumors arise mainly within the cerebellum and are of three principal types—known as medulloblastoma, astrocytoma and ependymoma.

The medulloblastoma is a malignant tumor which spreads along the subarachnoid spaces. It is impossible to extirpate it totally, and operative interference does not prolong the lives of these cases very much. An attempt to remove such a tumor serves therefore only to increase the operative mortality. These tumors are very radiosensitive but the effect of radiation does not persist beyond three to four years. Attempts to increase the amount of radiation result in severe degenerative changes in the brain itself. If one could make a sure pathologic diagnosis before exploration these tumors should not be operated upon. Recently Cutler, Sosman and Vaughan have proposed to radiate all tumors of the cerebellum in children. If the symptoms improve it would be concluded that the tumor was a medulloblastoma and no operation would be undertaken. I have not yet followed this plan, which was first proposed by Cushing, for fear that acute edema or hemorrhage might be provoked by the radiation and result in the death of a patient with a favorable tumor. Such deaths were not infrequent in the early days of radiation therapy before decompressions were made. But the suggestion is in the right direction of proposing a means of establishing before exploration a diagnosis of inoperable tumor.

The other frequent type of glioma in the cerebellum of children is the astrocytoma. It is a benign, avascular tumor, often cystic, which may be permanently removed with a very low mortality. It is not radiosensitive, hence the above suggestion for differential diagnosis.

The ependymoma is also a benign tumor but unfortunately is usually attached by a broad base to the floor of the fourth ventricle. Any attempt to dissect it free results almost invariably in edema and congestion of the bulb and death from respiratory failure. So my practice is to let it alone, as soon as a biopsy has established the nature of the tumor, and content myself with a decompression. It is unfortunately not possible at present to distinguish these ependymomas clinically from the astrocytomas or medulloblastomas.

We must mention another type of glioma of children which comprises about 15 per cent of all intracranial tumors at this age-group, that is the glioma pontis. These diffuse invasive tumors of the pons cause multiple palsies of the cranial nerves and, in spite of their proximity to the aqueduct of Sylvius, do not obstruct the circulation of the cerebrospinal fluid or cause intracranial hypertension until very late, if at all. In this way they may be recognized. They are, of

course, inoperable. Because of the lack of intracranial hypertension they are often not recognized but diagnosed encephalitis.

Again, as was the case with tumors in the region of the hypothalamus, we see how different would be the indications for operation could we only establish a pathologic diagnosis before operation. Only our ignorance leads us nearly always to perform an exploratory craniotomy, but our further procedure should vary with the nature of the tumor exposed.

In adults the most frequent cerebral tumor is the glioblastoma multiforme. It is an invasive, destructive, rapidly-growing tumor of the cerebral hemispheres. Few of these cases survive operation more than a year and none to my knowledge has ever been completely removed without recurrence. Autopsies show that the limits of these tumors are irregular, indefinite, so that even under the microscope it is difficult, often impossible, to identify their boundaries. Any surgeon who hopes to remove entirely such a tumor must be prepared to take away such a mass of tissue as seriously to cripple the patient. Even the cases reported in the literature of removal of a hemisphere must usually leave cells which have invaded the basal ganglia since these so-called hemidecerebrations are really hemidecortications. Also these tumors often spread through the corpus callosum into the opposite hemisphere. Although the surgical results are always disappointing, there are still numerous surgeons who advise an attempt to remove these tumors regardless of the mortality and morbidity. It seems to us that as long as no new methods of early diagnosis are perfected, or some new technic devised which offers hope, these tumors should be left alone, since experience has amply proven that they cannot be removed. Unfortunately the diagnosis usually remains in doubt until the cranium has been opened and biopsy made. If a preoperative diagnosis could be made, as in the case of the medulloblastoma, much useless operating could be avoided. Two methods have been proposed for the preoperative diagnosis of these tumors. Pfeiffer, also Creutzfeld, advise removing a cone of tissue through a trephine-opening as a method of biopsy examination. They claim that this can be done with small risk of fatality. Heretofore I have considered this method potentially too dangerous and have not attempted it. Recently Tönnis has claimed that glioblastomas can be diagnosed by arteriography. If his claim is verified we will have taken a long step in advance along the way which I have been advocating, since the method of arteriography is already relatively innocuous and will be constantly perfected (Gross).

Contrary to our experience with the medulloblastomas, roentgen radiation of glioblastomas has given only mediocre improvement

and has even sometimes proved fatal by provoking vascular accidents in the tumors. No better results can be obtained by increasing the dose of radiation since, as O'Connell and Brunschwig have shown, larger doses of radiation cause severe degeneration of the cerebral cortex. When a diagnosis of glioblastoma can be established the only reasonable surgical therapy is a decompression, and McKenzie argues even against that palliative procedure.

When the surgeon finds in the cerebral hemispheres an astrocytoma, an oligodendroglioma, or an ependymoma, an attempt to extirpate it is justified since these are relatively benign tumors and there is a better chance of total removal and permanent cure.

An attempt should always be made to extirpate the tumors known as meningiomas, that is to say the bulbous, encapsulated growths arising in the meninges. This is nearly always possible with greater or lesser risk depending on their accessibility and vascularity. Since they are encapsulated and push aside the brain-tissue, the latter recovers its function in large measure after the compression is removed. The principal danger is from hemorrhage, so preparations should be complete for immediate blood-transfusion whenever one of these tumors is encountered. It is particularly necessary in these cases for the operator to be prepared for a long and bloody struggle, hence the advisability of establishing a preoperative pathologic diagnosis.

It appears therefore to me to be of primordial importance to establish the pathologic nature of a tumor in the intracranial cavity before attacking it. But we must have methods of doing this. Certain presumptions based on the age of the patient, the location of the lesion, etc., put one on his guard. These indications I have developed in my book, *Intracranial Tumors*. But in this way one can rarely establish more than a probability. Ventriculography and encephalography localize tumors but rarely give any information concerning their nature. The ventriculographic findings of the medulloblastomas, astrocytomas and ependymomas in the region of the fourth ventricle may be identical but I have already indicated that they vary widely in operability.

There exists also a series of other findings which aid in establishing the nature of an intracranial tumor. The meningioma can often be recognized by certain characteristic changes which it produces in the overlying cranium. The craniopharyngioma is usually visible because of its calcification in the suprasellar region. The adenoma of the hypophysis causes a rather typical ballooning of the sella turcica. The glioma of the optic chiasm enlarges the optic foramina and is sometimes associated with the cutaneous manifestations of von Recklinghausen's disease. The oligodendrogliomas in the

cerebral hemispheres are the most common calcified tumors in this region in adults. The pinealoma causes a paralysis of upward conjugate movements of the eyes and is sometimes associated with a precocious puberty. The glioma of the pons causes numerous palsies of the cranial nerves and augmentation of the intracranial tension late or never. The neurinoma of the acoustic nerve enlarges the internal auditory meatus and destroys the labyrinthine responses on the same side. All of these signs enable us in certain cases to establish a pathologic diagnosis, but they may be missing and sources of error exist.

But it remains to differentiate the major part of the gliomas. Since I showed some years ago, with Cushing, that the different types of gliomas offered very different prognoses it has become very desirable to establish the type before operation. This is rarely possible. One may guess that he is dealing with an astrocytoma when a child has had symptoms of a cerebellar tumor for over a year. A calcified tumor in the cerebral hemispheres of an adult has a good chance to be an oligodendroglioma. But usually there is no clue either in the symptoms, history or findings.

I have already noted the suggestion of Cushing that all children who have symptoms of cerebellar tumor be subjected to roentgen radiation. If the symptoms improve one would conclude that the tumor is a medulloblastoma since that is the only type which is radiosensitive in that region during childhood. Experience is lacking to determine how accurate such a procedure might prove. Up to the present I have not adopted it for two reasons: (1) for fear of overlooking an operable tumor (one must remember that the effect of radiation on the medulloblastoma is not permanent) and (2) for fear of causing the death of some child from acute edema; such deaths were frequent in the early days of roentgen radiation when decompressions were not made before treatment. In spite of the fact that this suggestion is in line with the thesis of this address I have heretofore been too timorous to adopt it.

The same timidity has prevented me from following the procedure of Pfeiffer and Creutzfeld for the diagnosis of cerebral tumor in adults. They use the old method of brain-puncture with a sharp, cylindrical, hollow needle devised by Neisser and Pollak. The cone of tissue removed is smeared and examined for neoplastic cells. I have not adopted this method for fear of hemorrhage. These authors admit several deaths from this source. It might be possible to recognize glioblastomas without much difficulty from the tissue removed; Eisenhardt and Cushing have explored the possibilities of pathologic diagnosis from such smears of biopsy tissue. Recently Tönnis has maintained that it is possible to recognize glio-

blastomas from the appearance of x-ray photographs made after injection of thorotrast into the carotid artery. This method of arteriography devised by Moniz of Lisbon is relatively innocuous and, if Tönnis's contention is confirmed, a great step in advance will have been made in the differential diagnosis of cerebral tumors.

Until these newer methods are assessed and established, or others devised, we shall continue to be obliged to make explorations and depend upon biopsies, made when the tumor is exposed, to determine its nature. One may make a histologic examination by the supravital method as advised by Cushing and Eisenhardt but, after long experience, I prefer to make a frozen section since the architecture of the growth is important for the diagnosis and this may be lost in a smear. Whenever the diagnosis of glioblastoma or medulloblastoma is established I make no attempt to remove the tumor. If it is a medulloblastoma I make a decompression and subject the tumor to roentgen radiation. If the tumor is a glioblastoma I make also a decompression and radiate, but without much hope of improvement. Some surgeons, McKenzie for example, in such a case will not hesitate to remove sufficient tumor, or even normal brain-tissue, to permit firm closure of the skull in order to avoid herniation and possible fungus cerebri. The patient's symptoms are then relieved by drugs.

The operative indications for intracranial tumors are therefore logically to be derived from their pathologic study if we take into account the deficiencies in our diagnostic skill. We must consider also the tissue in which the tumor develops in planning the surgical intervention. Abstention from operation if the diagnosis can be established, or palliative procedure if exploration is necessary for diagnosis, are indicated for malignant tumors such as glioblastomas and medulloblastomas; radical excision is indicated for benign tumors such as the cerebellar astrocytomas. In the treatment of malignant tumors removal of large masses of nervous tissue should not be condoned, but if necessary to gain access to a benign tumor it may in certain cases be justifiable. We must direct our efforts in the future toward the solution of the problem of preoperative pathologic diagnosis. The situation calls for temerity. Our thinking in regard to this problem in the past has been as mixed up as Ned Brace's terrapin.

SURGICAL PROBLEMS PRESENTED BY JAUNDICE IN INFANCY AND CHILDHOOD

STANLEY J. SEEGER, M. D.
Milwaukee

THE fact that surgical intervention is to be seriously considered in infancy and childhood in several conditions in which jaundice occurs makes advisable a discussion of the problems which this sign presents. Jaundice is common in infancy and like cyanosis and albuminuria it is of varying significance. It is a condition brought about by the presence of bile pigment in the blood which produces a yellowish discoloration of the skin, sclera and mucous membranes. The brain, with the exception of certain areas, the spinal cord and cartilage do not become bile-stained no matter how intense the general discoloration may be. No theory completely explains the variations in staining of tissues. Some authors believe that the staining of a specific tissue is related to its function as regards the formation and excretion of bilirubin. The primary pathologic condition in every case of jaundice is the presence of an excessive amount of bilirubin in the circulating blood. It is well known that discoloration of the sclera and skin may be overlooked if light of a proper character is not available, but the frequency with which milder degrees of jaundice are missed in clinical examinations is quite apparent in the review of any large number of cases. The blood normally contains 0.5 mg. per cent of bilirubin and 2 mg. per cent will give clinical jaundice. The normal icterus index is from 4 to 6 and jaundice should be noted clinically when the icterus index approaches 20. It is not uncommon to see patients with an icterus index of from 30 to 35 in whom clinical jaundice can be observed only on careful inspection in a good light. In determining the degree of jaundice the bilirubin content of the blood is a more accurate measure than is the icterus index.

It is generally accepted that bile pigment is derived solely from the breaking down of hemoglobin and that this destruction results from the activity of the reticulo-endothelial system. The principal sites of the formation of bilirubin are the bone marrow, the spleen and the liver. In the liver this production takes place as a result of the action of the Kupffer cells and not as a result of the activity of the polygonal cells. The typical liver cell in relation to bilirubin serves only as an excretory cell. There are, as a result of this physiologic mechanism, three possible causes of jaundice:

1. Bilirubin is normally produced, taken up by the liver cells, excreted in the bile and meets obstruction to its normal passage into the duodenum.

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

2. Bilirubin is formed normally, but owing to damage or functional derangement of liver cells it may be unable to enter them and the bile goes directly into the circulation. The variation of this difficulty takes place when the cells are normal but an excessive amount of bilirubin is formed and brought to them for excretion.

3. A combination of damage to polygonal cells and obstruction of the bile passages. The deficiency and derangement of the liver cells may be caused by a toxic agent, either non-bacterial or bacterial.

The van den Bergh reaction is of significance in the differential diagnosis of types of jaundice. If the serum contains bile which gives a direct reaction it means that the bilirubin has passed through the polygonal cells. According to Astrachan the direct reaction always indicates disruption of bile canaliculi whether this results from obstruction or is secondary to necrosis of the liver cells. A delayed direct or indirect reaction is given by serum in which the bilirubin has not been through the polygonal cells, the classic example being the hemolytic types of jaundice. Some clinicians believe that an indirect reaction in serum containing bilirubin in amounts more than normal does not always mean hemolytic disease and in fact may definitely point to associated hepatic injury. Both types of serums give an indirect reaction, the difference being that when serum bilirubin gives a prompt direct reaction the indirect is not resorted to except for quantitative determinations. When the direct reaction is present the bilirubin is excreted by the kidneys when the concentration in the blood is greater than 1 part per 50,000. When the indirect reaction alone is present bilirubin does not pass through the kidneys. The latter type of jaundice is referred to as acholuric jaundice. While a consideration of the disturbed physiology seems to simplify the classification of jaundice for clinical purposes, the problems presented are difficult of solution because of the limitations of our knowledge and because one often encounters a combination of conditions.

In infancy and childhood three conditions in which jaundice is a presenting sign are of particular interest to the surgeon:

1. Congenital anomalies of the extrabiliary ducts,
2. Erythroblastosis foetalis, and
3. Hemolytic jaundice of the so-called congenital or familial type.

Congenital obliteration of the extrahepatic bile ducts is not a rare condition. In 1916 Holmes had collected over 100 cases from the literature and since that time numerous references to its occurrence have been made. In 1935 Ladd reported 45 cases, and at Milwaukee Children's Hospital six cases have been classified under this diagnosis.

The liver in its development may be regarded as a direct extension of the foregut. The ducts within the liver, like those of any other

gland, arise by a secondary process from undifferentiated tissue lying along the distribution of the portal vein. These cells arrange themselves in cords and develop lumina. The extrahepatic ducts arise within the solid liver outgrowths. At first the gallbladder lies in the ventral mesentery or gastrohepatic omentum or ligament, a position which is permanent in some vertebrates, and which may occur as an anomaly in man. In the second month it becomes imbedded in hepatic tissue, and it assumes its superficial position only at a later date. The lumen of the gallbladder and the ducts is occluded by an epithelial proliferation during the second month of intrauterine life just as is the lumen of the duodenum. Some authors ascribe occlusions of the ducts to failure of these epithelial plugs to disappear. It should be pointed out in this connection that the condition classified as congenital obliteration of the bile ducts is not as simple as one might picture it if one had in mind only an obstructive process or a malformation localized to a segment of the extrabiliary tract. Some students of this subject, notably Rolleston, are of the opinion that in most of these cases there is primarily a mixed cirrhosis of the liver, which gives rise to a descending obliterative cholangitis. Hepatic cirrhosis is common in congenital obliteration of the bile ducts and is not accompanied by dilatation of the intrahepatic ducts such as is seen in chronic obstruction of the common duct in the adult. In our cases which have come to autopsy there has been present in all a hypoplasia of the bile ducts with marked stasis of bile. Holmes reported a case in which careful serial sections of the biliary tract showed the common duct entirely replaced by loose connective tissue. While a great variety of lesions are found, from a clinical surgical standpoint only those involving the common duct or the hepatic ducts are of importance. In half of the reported cases in which the common duct is affected there are associated lesions, commonly an imperfect cystic duct or malformation of the hepatic ducts or both. In over 16 per cent of all reported cases of atresia of the bile ducts both the cystic and the hepatic ducts were essentially normal and communicated with each other. In Ladd's series of 45 cases 15 were theoretically amenable to surgical relief. When the extrahepatic arrangement is such that establishment of a communication between the gallbladder and intestine will apparently permit the free drainage of bile the possibility of the existence of extensive intrahepatic insufficiency should not be overlooked. As a rule the liver is enlarged in these cases. It is bile-stained, often dark green and in many instances is described as purple in appearance as was true in several of our cases.

In the differential diagnosis of obstructive jaundice due to a defect of the bile ducts one must consider icterus neonatorum simplex which occurs in from 30 to 80 per cent of all newly born infants. This

condition rarely occurs later than the fourth day and usually lasts for from two days to two months. In icterus neonatorum the vomitus and the stools contain bile and the van den Bergh reaction is of the indirect type. When the jaundice lasts longer than two weeks and when the severity has a tendency to increase one should suspect that a more serious condition exists. Syphilis and sepsis should be ruled out as possible causes but in the absence of these conditions the probability of obstructive jaundice must be considered. The absence of bile or urobilinogen in the stools or in the duodenal contents, the presence of bile in the urine, high icterus index, high bilirubin content of the blood, a positive direct van den Bergh, and an enlarged liver all indicate an obstructive lesion involving the bile ducts. In severe cases of obstructive jaundice, at times the stools may show some evidence of yellowish discoloration. This may be due to contamination from the urine or from the passage of bilirubin through the intensely stained cells lining the intestinal tract.

The tendency to hemorrhage in jaundice is well known and in this connection the classic work of Quick on prothrombin and liver function should be noted. The recent advances in our knowledge concerning the hemorrhagic tendency in jaundice and its importance in surgery cannot be discussed at this time. In these patients the spleen is usually moderately enlarged. The fact that some of these children live for nearly a year with complete biliary obstruction is commented on by several authors and the nutrition is sometimes quite well maintained.

A few reports of spontaneous recovery, both complete and partial, are to be found in the literature. One case of spontaneous recovery from obstructive jaundice due to defective extrabiliary tracts is reported here. The lesion may be incomplete or partial and accessory passages may be present.

Calculi have been reported present at birth but in a series of 851 autopsies at the Milwaukee Children's Hospital no calculi have been found.

Time aids one in making a final diagnosis of congenital biliary obstruction. One can well wait until the infant is a month old before making a final decision, but Ladd emphasizes the necessity of operating on these patients before the end of the second month because of the danger of secondary changes as a result of the continued obstruction and jaundice. In operating on these patients one should be sure that the gallbladder communicates with the common duct and the liver before making an anastomosis between it and the intestine. Ladd has made the valuable suggestion that a catheter be inserted into the gallbladder and that through it normal saline solution be introduced under a mild degree of pressure. In this way

the cystic and common ducts can sometimes be distended, and both Ladd and Penberthy report instances in which a plug of inspissated bile has been released from the lower portion of the common duct by this maneuver with the result that the obstruction has been completely relieved. Except in the rare instances in which spontaneous cure has occurred the condition is inevitably fatal unless the obstruction is amenable to surgical relief.

Fig. 1. Photomicrograph of normal liver showing typical periportal space.

The following cases from the Milwaukee Children's Hospital have been classified as congenital anomalies of the bile tracts:

CASE 1.—A female child, born at term Jan. 27, 1927, was admitted April 11, 1927. The Wassermann test was positive. Marked jaundice of the obstructive type was noted. The liver was palpable two fingers below the costal margin. Congenital syphilis and congenital obliteration of the bile ducts were diagnosed. She died suddenly on April 15.

The post mortem revealed a normal gallbladder, which however did not contain bile. There was neither cystic duct nor common duct, both being replaced by fibrous tissue. The hepatic ducts were apparently incomplete. The liver showed lack of definite structure. There were strands of liver cells isolated by fibrous tissue in which there were leukocytes and deposits of bile. There was hypoplasia of the bile ducts.

This case is of interest since it presents the complication of two conditions, either of which might account for marked jaundice.

CASE 2.—A female child was born April 7, 1935. Five weeks later she was admitted to the hospital. Apparently she had become jaundiced soon after birth. The abdomen was distended on admission. Duodenal suction relieved the distention. She vomited frequently. The stools were clay-colored. The jaundice was of the obstructive type.

At operation a large amount of bile-stained peritoneal fluid was found. The intestines were matted together. The common duct and hepatic ducts were not visualized. A small amount of bile-stained fluid drained on several occa-

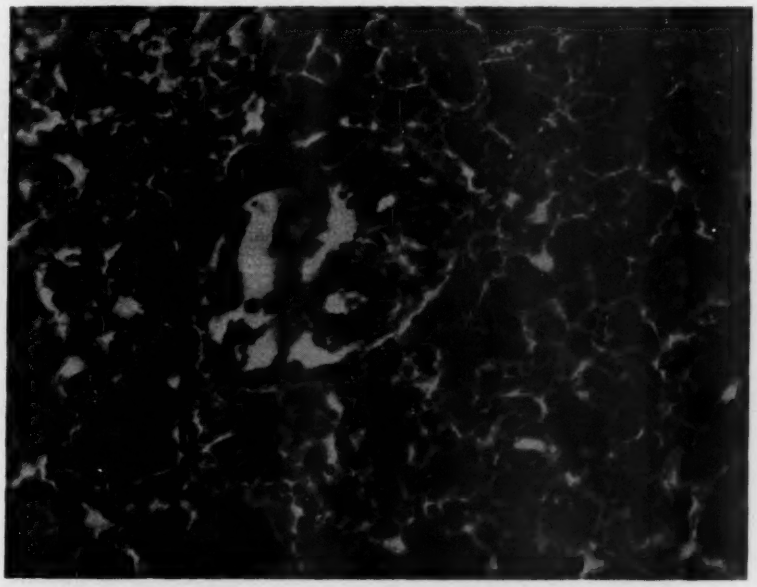

Fig. 2. Photomicrograph of liver from typical case of congenital atresia of the bile ducts. (Case 2.) This section shows a periportal space with an artery comparable in size to that shown in picture of normal liver (Fig. 1). The bile duct is poorly developed, the liver cords are distorted and hepatitis is definite.

sions but there was no bile drainage from the catheter. The child died on June 21, three weeks after operation.

Post mortem showed obliteration of the lower portion of the common duct and a perforation of the short patent portion. The hepatic ducts were obliterated. There was generalized peritonitis. There was marked congestion of the liver. The sinusoids were distended with red blood cells. Many of the liver cells, especially in the periportal area, contained large amounts of brownish bile pigment. There were numerous areas of miliary focal necrosis in which the nuclei were lost. There was marked hypoplasia of the intrahepatic bile ducts and bile stasis.

This case is of interest as it illustrates the possible extension of hepatitis to the peritoneal cavity. The question of latent sepsis as a causative factor in the production of this condition should also be considered.

CASE 3.—A female child, jaundiced since birth July 17, 1935, was admitted Jan. 13, 1936. At times the jaundice had seemed to fade and the stools at these times would appear somewhat darker. The stools were light when the child was jaundiced. She had weighed 14 pounds at 5 months. One week before admission this patient was taken sick with a cold, fever, cough and loss in weight. On admission there was marked jaundice of the obstructive type. The liver was 2 cm. below the costal margin. There were signs of fluid in the peritoneal cavity. Four hundred eighty cubic centimeters of fluid were aspirated on Jan. 14, 1936. This fluid contained gram positive cocci and bacilli. Icterus index was 200. White blood cells were between 35,000 and 53,000. This was a positive direct van den Bergh. The patient was tapped several times. On January 21, 500 c.c. of dark olive green fluid was obtained and on the 31st mucoid material was drained off.

On February 10 the abdomen was opened under local anesthesia. The liver was large, greenish in color and its lobulations were abnormal. There were several small projections of liver tissue near the fissure. No free fluid was in the abdomen. There were many adhesions over the right lobe of the liver which was adherent to the abdominal wall. The gallbladder was small, thick-walled. On opening the gallbladder a large amount of bile-stained purulent material escaped. Forceps dropped 10 cm. into the gallbladder and gave the impression that an abscess might have perforated. Thick, cloudy bile flowed from the abdomen. The patient died within 24 hours. No post mortem was done. Pneumococci were grown from the fluid.

This case also raises the question of sepsis as the cause of hepatitis and cholangitis with a resulting secondary obstruction of the ducts, even though the sepsis was not evident from the external appearance of the patient nor was any infection of the navel noted at the time of birth. Amberg has stated that exacerbations of hepatitis in infants may be mistaken for respiratory infections.

CASE 4.—A female child aged 5 weeks was admitted on Feb. 23, 1937, because of cerebral symptoms probably due to hemorrhage into the ventricles and subarachnoid spaces. She had been jaundiced since birth. Signs and symptoms suggested an acute hydrocephalus. Hemorrhage was thought to be secondary to some underlying blood dyscrasia or to jaundice. The tendency to develop hydrocephalus ceased after three weeks. The liver was large, and extended down to the umbilicus. Stools were clay-colored. The urine contained bile. The direct van den Bergh was positive. Icterus index was 100.

Operation was done March 27 at the age of 9 weeks. The gallbladder was small, 1 cm. in diameter and contained no bile. The cystic duct was patent for a short distance, but contained no bile and a probe could not be passed into the duodenum or upward. Both hepatic ducts were obliterated. A tube was placed in the gallbladder but no bile drained. The patient died on April 8, following disruption of the wound.

Post mortem showed, in addition to the condition noted at the time of operation, a hypoplasia of the intrahepatic bile ducts and marked bile stasis.

CASE 5.—A male child, born Nov. 24, 1936, was admitted Dec. 28, 1936, with typical signs of obstructive jaundice. He had been jaundiced since a week after birth. Icterus index ranged from 35 to 45. Van den Bergh direct was immediate. Stools showed no urobilinogen. The liver was 1 finger-breadth below the costal margin. The spleen was not palpable.

At operation on January 18 the gallbladder was found to be small and empty. It was opened near the ampulla. The cystic duct was followed to the junction with the common duct. It was patent to this point. The common duct was small, contained no bile and apparently only a short segment was open. A probe could not be passed upward into the hepatic ducts, both of which were obliterated and there was no opening downward into the duodenum. The exposure was satisfactory. The hepatic artery and portal vein could both be identified, but apparently the ducts were missing. The liver was congested and reddish green. The spleen was slightly enlarged. A Penrose drain was placed down to the common duct.

There was a very slight greenish discharge on the dressing on one occasion but no bile drainage from the wound at any time. The parents were told that this child could not survive. There was no urobilin or urobilinogen in the stools on July 7, six months after the operation, and the icterus index was 35 and the van den Bergh was positive direct. It was of interest that on two occasions (March 12 and March 18) after four months of complete obstruction the prothrombin content was 100 per cent, this having been done by Quick. On July 29, in the sixth month after operation, urobilinogen appeared in the stools, the jaundice fading. Within a month the icterus index dropped to 8 and the van den Bergh reaction disappeared.

This child has not been jaundiced since. He has developed normally. His liver is still two fingerbreadths below the costal margin and the spleen is palpable. He was last seen in January, 1940, at which time there was no evidence of jaundice.

In this case it is obvious that simple plugging of the ducts by inspissated bile was not the cause of the obstruction. It is probable that the epithelial proliferation mentioned by Keith as occurring during the second month of intrauterine life persisted and that canalization of the ducts was delayed.

CASE 6.—A female child, born May 25, 1937, was admitted at the age of 3 months with a high fever. Her birth weight had been 4 pounds 12 ounces. On admission she was poorly developed and poorly nourished, rachitic and jaundiced, weighing 6 pounds 12 ounces. Icterus index was from 58 to 60. Van den Bergh showed moderate direct reaction. No history of infection of the umbilicus at birth. At no time did the stools show any urobilinogen. There was moderate secondary anemia and increased fragility of the red blood cells.

Her condition remained generally poor and for this reason operation seemed contraindicated. X-ray examination of the gallbladder following dye administration showed a non-functioning gallbladder. She died October 28. No autopsy was permitted.

This case in all probability was one of congenital abnormalities of the bile ducts. The fever suggests the possibility of hepatitis as an important factor in this case.

The second group of cases in which surgical intervention should be considered are those which are classified as icterus gravis or erythroblastosis neonatorum. Erythroblastosis neonatorum is considered as a toxic anemia because of the changes in the appearance of the blood and the pathologic changes in the tissues. Several forms of the same condition are described under different names, the name

being chosen from the most prominent characteristic. The severity of the condition varies with the different forms.

It is generally accepted now that the more commonly known idiopathic congenital anemia of the newborn is the lightest form of this condition and that erythroblastosis foetalis, icterus gravis and universal edema are very much more severe variations. The so-called aregenerative edema of the newborn is also considered a light form of erythroblastosis. The disease is often first suspected at the delivery table with the delivery of an infant with a yellow stained vernix and an enlarged placenta. The stain of the vernix and enlargement of the placenta are not pathognomonic but are often present with the disease.

The infant may be jaundiced or edematous on delivery, but if not, he will become rapidly and progressively jaundiced within a few hours. Usually the cry is weak and there may be nervous symptoms and convulsions in more severe forms. The condition is familial, and as far as is known, never occurs in the first child. Once the disease has become manifest it can be said with fair certainty that some form will be seen in the subsequent children of that mother. Toxicity of any form in the mother is especially rare and is considered part of another process, if existing, and the deliveries themselves are generally without difficulties.

When the disease is suspected a blood count will show a profound anemia, the red count usually being about 1,000,000. The white count will be high both from the leukocytosis, which is present and, because of the large numbers of immature red forms not laked by acetic acid. The differential count shows a great many of all forms of immature red blood cells (erythroblasts, megaloblasts and normoblasts). There will be, also, all forms of immature white cells with usually a great number of eosinophils and eosinophilic myelocytes. Reticulocytes usually are found in greater numbers than in most any other condition. The anemia is macrocytic with many appearances in the red blood cells typical of pernicious anemia (changes in size, shape, staining and stippling). The number of immature red blood cells is over 5,000 per cubic millimeter and very often over 100,000. The color index is often high.

The icterus index is elevated and in the universal edema form of the condition it may be as high as 15 with no clinically recognizable jaundice. Gastrointestinal hemorrhages are common. The fragility test is normal and of no apparent importance. The albumin-globulin ratio is normal even in cases with edema. The Wassermann and tuberculin tests and blood cultures are not of significance. No nationality apparently is spared. Bleeding and coagulation time are normal and sometimes prolonged. Liver and spleen are also en-

larged though the spleen may not be palpable until after one or more weeks in less severe forms.

Clifford and Hertig report the disease in 7 out of 2,400 infants delivered, and Hawksley and Lightwood, Diamond and others report about the same incidence. Aside from the staining of tissues and edema found in some forms, petechiae and ecchymoses are found commonly in the organs. Some enlargement of the heart and a form of hemorrhage in the lungs having a lobular distribution which is called "hemorrhagic bronchopneumonia" are often present.

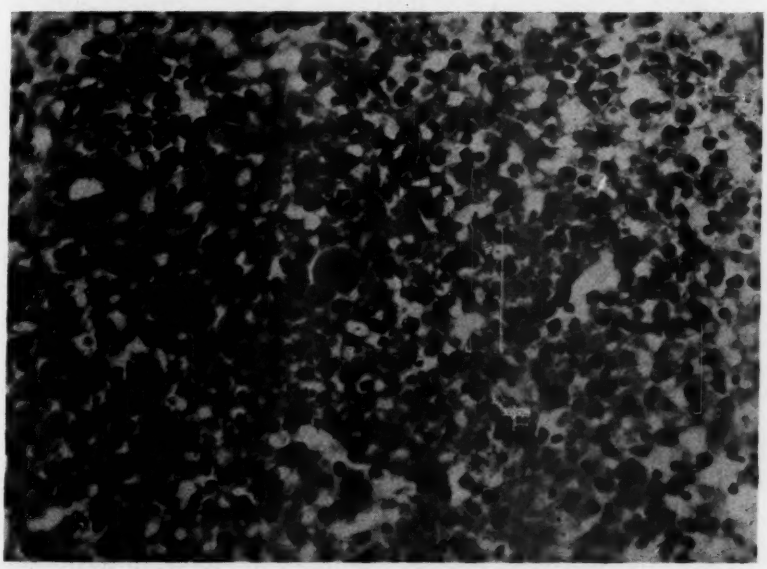

Fig. 3. Photomicrograph of spleen from case of erythroblastosis foetalis showing typical area of erythrogenesis.

True bronchopneumonia may be terminal. The liver and spleen are often very large and are of a deep cherry red brown color due to the hemolysis and hematopoiesis. The spleen may not be palpable for one or two weeks in milder forms. The liver and spleen, and sometimes the pancreas, adrenal and thyroid, have large islands of hematogenesis with atrophy of liver cords. In old cases some claim to have found young fibroblasts. All young forms of red and white cells are seen with eosinophilic forms very numerous. The spleen is engorged with blood, has islands of hemogenesis, large numbers of eosinophils and there is much blood pigment throughout the organs from the hemolysis.

The brain shows anoxemic degeneration, commonly in the nuclear areas. Schmorl describes a more circumscribed form of degen-

eration which he calls "kernikterus" that is also common in the basal ganglia. He found this in 6 out of 120 cases and spontaneous cranial hemorrhages are found in about 1 out of every 3 cases. These areas are always stained intensely with bilirubin or its products of oxidation.

The importance of erythroblastosis neonatorum to the surgeon lies in the fact that splenectomy seems to offer some hope of relieving the condition. Cooley, in 1932, advocated this operation and stated that not only did splenectomy relieve the child of a large mass but in his opinion the progress of the disease would be arrested. He reported operating on four patients, one of whom died one week after operation due to a streptococcic throat infection. Another died five years later of septicemia having lived comfortably during the intervening time. At the time of his reports two patients were still alive, the disease apparently having been arrested. Penberthy has reported success following splenectomy in two cases. Hitzerot has reported four cases in which the spleen was successfully removed in this condition and in which the patients survived for varying periods of time up to 18 years. The blood picture of erythroblastosis continued but death in all instances was due to some intercurrent infection.

The generally accepted therapeutic measure is blood transfusion. The argument that the liver may be overloaded by extra cells is not considered important by Tow. He also emphasizes the importance of changing donors if the improvement does not follow the first or second transfusion. He maintains that there may be a factor in some donors which stops hemorrhage. This condition is one form of the disease, the milder forms of which recover with treatment by transfusion or without treatment, the severe forms being highly fatal or developing the so-called congenital foetalis hydrops or universal edema. When transfusion does not halt the hemolytic process splenectomy should be resorted to and the importance of giving consideration to this procedure at an early stage should be emphasized.

At Columbia Hospital four cases of erythroblastosis foetalis occurred in 1,618 infants, in 1,609 deliveries. One infant was still born. The condition was of a fulminating type in the three other cases, the patients living from 13 hours to three days. Transfusions had no apparent effect. At Milwaukee Children's Hospital six cases have been classified under the diagnosis of icterus gravis and erythroblastosis foetalis. Two of these patients died on the day of admission. One died on the third day in the hospital. Three recovered, all who recovered having had repeated transfusions. Splenectomy was not done in any of the ten cases here reported.

The third group of cases in which jaundice is an associated sign is the so-called hemolytic icterus of the familial or congenital type. In this condition jaundice is usually present at birth or soon afterward and varies in intensity from time to time. These variations seem to depend somewhat upon recurring disturbances, among these being the respiratory and other infections or digestive disturbances. During acute exacerbations the spleen is enlarged and it recedes after the crisis has passed. At these times tenderness and pain may be present over the spleen. Gallstones occur with great frequency in these patients, probably as a result of the marked concentration of the bile. At times the jaundice may be easily overlooked. In a recent case with an icterus index of 30 it was necessary to scrutinize the sclera carefully in the bright daylight in order to detect the icteric tinge.

The important laboratory findings in this condition are the presence of bile in the stools, its absence from the urine, the increased fragility of the red cells, the presence of an indirect van den Bergh reaction, the absence of a direct van den Bergh reaction and the increase in the icterus index and of the bilirubin content of the blood. Morphologic changes in the red cells are also present. During the crises there is often a very marked anemia, and reticulocytes are commonly found. The disease does not shorten life but, as Rolleston says, while patients may not be really ill, they are not often well. The recurrence of crises with marked anemia, a splenic tumor and the development of gallstones all demand adequate therapy. Splenectomy in this condition is generally accepted as the classic treatment.

In addition to the three general groupings of patients with jaundice in which surgical intervention should be considered we have encountered some unusual forms, several of which merit mention. Jaundice has been associated with hypertrophic pyloric stenosis in several instances. In these cases the condition was a prolonged form of icterus neonatorum which did not interfere with the surgical procedure and which cleared up immediately following surgery. One patient, a boy aged 10, was admitted with a history of pain about the umbilicus of three weeks' duration. Jaundice was noticed soon after the onset of pain. No nausea or vomiting had occurred. He was markedly constipated. The stools were clay-colored. The condition was afebrile from the onset and on admission to the hospital, at the beginning of the fourth week of symptoms, the temperature was normal and the white count 9,200. The referring physician suspected a malignant tumor involving the common duct. On exploration of the abdomen a large mass of edematous lymph glands with marked inflammatory reaction about the lesser omentum was found. There were no stones in the gallbladder or common duct.

The gallbladder was large and contained thick inspissated bile. Its walls were thick and edematous. The abdomen contained clear bile stained fluid. The appendix was bound down in the midline by adhesions. Because of the extent of the inflammatory reaction and the large size of the glands in the gastrohepatic omentum, a cholecystgastrostomy was performed. The appendix was also removed. Convalescence and subsequent course were uneventful. The jaundice cleared up without any unusual delay. The pathologist's report on the lymph gland showed a chronic hyperplastic lymphadenitis with no evidence of tumor formation. The appendix was subacutely inflamed. Another boy, aged 11 years, was admitted with a history of having been struck in the abdomen by a playmate four days before admission. That night he had pain in the upper right quadrant of the abdomen. He vomited several times on the first and fourth days. The pain radiated to the left upper quadrant. On admission four days later he was jaundiced. The abdomen was tense in the upper portion and tender. There was bile in the urine. The stools showed a very faint trace of bile and were slate colored. The icterus index was 100. The van den Bergh showed a moderate direct reaction. The blood otherwise was negative. He had no fever. Within a week the jaundice began to disappear and on discharge, 12 days after admission, the stools were normal in color, and the jaundice had practically disappeared. There was a small mass in the upper right quadrant which was thought to be a hematoma in the rectus muscle which subsequently disappeared. This case was probably one of obstruction of the common duct due to hemorrhage. Toxic infectious jaundice, which should always be differentiated from other types, is illustrated by the case of an infant born May 20, 1929, and admitted June 21, 1929, with extensive erysipelatous infection over the abdomen apparently beginning near the umbilicus and involving the chest with marked jaundice. This patient died on June 29, 1929, the diagnosis of the cause of jaundice being obvious.

BIBLIOGRAPHY

1. Amberg, S., and Walters, W.: Infantile Jaundice: Report of Two Unusual Cases in which Cholecystostomy was Employed, Proc. Staff Meet., Mayo Clin. *13:* 769 (Dec. 7) 1938.
2. Astrachan, M.: Jaundice in Children; Report of 6 Cases of Icterus Gravis Neonatorum, Am. J. Dis. Child. *53:* 137 (Jan., pt. 1); 541 (Feb.) 1937.
3. Braid, F., and Ebbs, J. H.: Atrophic Cirrhosis of Liver Following Icterus Gravis Neonatorum with Pathological Report, Arch. Dis. Childhood *12:* 389 (Dec.) 1937.
4. Bushnell, L. F., and Aldrich, C. A.: Erythroblastosis Foetalis: Report of Case in which Patient was Treated and in which there were Clinical Signs of Kernicterus without Postmortem Confirmation, Am. J. Dis. Child. *53:* 1053 (April) 1937.
5. Cooley, T. B., and Lee, P.: Erythroblastic Anemia, Am. J. Dis. Child. *43:* 705, 1932.
6. Diamond, L. K.: Indications for Splenectomy in Children, Results in 52 Operated Cases, Am. J. Surg. *30:* 400 (Feb.) 1938.
7. Diamond, L. K.; Blackfan, K. D., and Baty, J. M.: Erythroblastosis Foetalis and

its Association with Universal Edema of Fetus, Icterus Gravis Neonatorum and Anemia of Newborn, J. Pediat. *1:* 269, 1932.

8. Dods, L.: Indications for Splenectomy in Pediatric Practice, M. J. Australia *1:* 377 (Feb. 26) 1938.
9. Holmes, J. B.: Congenital Obliteration of the Bile Ducts; Diagnosis and Suggestions for Treatment, Am. J. Dis. Child. *2:* 405, 1916.
10. Hoover, F. C., and Blankenhorn, M. A.: Dissociated Jaundice, Arch. Int. Med. *18:* 289, 1916.
11. Keith, A.: A Demonstration on Constrictions and Occlusions of the Alimentary Tract of Congenital or Obscure Origin, Brit. M. J. *1:* 301, 1910.
12. Keith, A.: Human Embryology and Morphology, Ed. 5. Baltimore: William Woods & Company, 1933.
13. Ladd, W. E.: Congenital Obstruction of the Bile Ducts, Ann. Surg. *102:* 742, 1935.
14. Macklin, M. T.: Erythroblastosis Foetalis (Icterus Gravis, Hydrops Foetalis and Congenital Anemia): Study of Mode of Inheritance, Am. J. Dis. Child. *53:* 1245 (May) 1937.
15. McMaster, P. S., and Rous, P.: The Biliary Obstruction Required to Produce Jaundice, J. Exper. Med. *33:* 731, 1921.
16. McNee, J. W.: Jaundice: Review of Recent Work, Quart. J. Med. *16:* 390, 1923.
17. Parsons, L. G.; Hawksley, J. C., and Gittins, R.: The Hemolytic Erythronoclastic) Anemias of the Neonatal Period with Special Reference to Erythroblastosis of the Newborn, Arch. Dis. Child. *8:* 159, 1933.
18. Penberthy, G. C., and Benson, C. D.: Surgery of the Biliary Tract in Infants and Children, Am. J. Surg. *40:* 232 (April) 1938.
19. Penberthy, G. C., and Cooley, T. B.: Results of Splenectomy in Childhood, Ann. Surg. *102:* 645, 1935.
20. Potter, A. H.: Gallbladder Disease in Young Subjects, Surg., Gynec. & Obst. *46:* 795, 1928.
21. Quick, A. J.: Nature of Bleeding in Jaundice, J. A. M. A. *110:* 1658 (May 14) 1938.
22. Reid, M. R., and Montgomery, J. C.: Acute Cholecystitis in Children as a Complication of Typhoid Fever, Bull. Johns Hopkins Hosp. *31:* 7, 1920.
23. Rolleston, H., and McNee, J. W.: Diseases of the Liver, Gallbladder and Bile Ducts, Ed. 3. London: The Macmillan Company, 1929.
24. Rosendaal, H. M., and others: Slight and Latent Jaundice, J. A. M. A. *104:* 374, 1935.
25. Skemp, A. A.: Gallstones in Infancy, J. A. M. A. *96:* 108, 1931.
26. Tow, A.: Anemia of the Newborn, Arch. Pediat. *55:* 745 (Dec.) 1938.
27. Yllpo, A.: Icterus Neonatorum und Gallenforbstoffsekretion beim Foetus und Neugenbrenen, Ztschr. f. Kinder j. *60:* 2161, 1913.

NAILING OF THE FRACTURED NECK OF THE FEMUR WITHOUT X-RAY

WILHELM ZUELZER, M. D.
Dallas

INTERNAL fixation for fractures of the neck of the femur is now the generally accepted treatment. Encouraging end-results, based on studies of cases over long periods of time, are being reported from various sections of the country.

One of the major difficulties of this treatment is the proper placing of the nails or screws. Many new methods to meet this issue have been presented in the recent literature. Most of them are based on mechanical guides, accessory table tops, and other similar appliances designed to simplify the procedure. Despite the mechanical aids the usual method is complicated and is practical only in the hands of skilful surgeons in well equipped hospitals.

Roentgenographic control by anteroposterior and lateral plates is essential in practically all of the advocated procedures. There are several disadvantages associated with the use of x-rays during the operation. It is difficult to procure satisfactory lateral views; the aseptic operating conditions are always endangered and the operation is prolonged, consequently exposing the soft tissues and bone a relatively long period. Due to the delay, if local anesthesia is used, its effect may be lost. And even the x-ray might deceive us. In a recent article King and his collaborators stress the importance of accurate roentgenography which allows correct interpretation. Especially misleading is the "flexed thigh position" to obtain the lateral view.

Only a few operators work without roentgenographic aid during the operation and they admit that poor nail placements frequently result. The slightest mistake in the direction, when the nail is inserted, produces a marked deviation in its course when it is driven up through the neck into the femoral head. But next to an accurate reduction, the most important step of this treatment is the exact placing of the nail. The handle increases the length of the nail furthermore complicating its accurate insertion.

At the present time there is a need for a more simplified method of internal fixation for fractured femoral necks. It should be easy to use, applicable to varied types of fractures and patients, without endangering complications, and capable of consistently producing an accurate insertion. It is the belief of the author that the mechanical guide, described below, meets these requirements.

Correct reduction of the fracture has to be accomplished before operative fixation can be considered. I prefer the use of a simple

Jones splint which gives traction, abduction and internal rotation. The principle is similar to the Roger Anderson splint.

Operative Procedure

After the usual lateral incision the prongs of the instrument are introduced anteriorly and posteriorly below the greater trochanter.

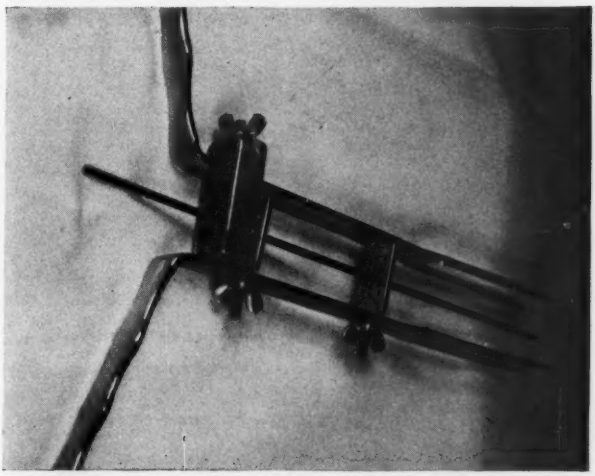

Fig. 1. The mechanical guide. Note the guiding wire in the center. One bigger opening admits directly Smith-Petersen nail.

In the same relationship close to the bone they are passed upward to the femoral neck and head (fig. 1, 2 and 3). The prongs are not bigger than medium-sized aspirating needles. The direction of the prongs is previously determined by an anteroposterior x-ray. Two of the prongs lie anterior to, and two posterior to the bone, all being laterally connected to each other. After their introduction they are tightened against the bone by means of a set of screws. These four prongs prevent any rotation or displacement of the fragments during the operative procedure. The accurate position of the two prongs anterior to the trochanter, neck and head can be checked with the index finger. The operator is now able to insert directly the Smith-Petersen nail or the guiding wire in the center parallel to the two prongs in front. In addition it is possible to control the insertion of several wires. By means of this mechanical guide, fixed both anteriorly and posteriorly to the bone, correct direction in placing the nail is assured. Also the necessity for frequent x-ray checks is obviated.

As stated above, an anteroposterior x-ray is necessary before the operation. Several wires attached anteriorly to the skin (fig. 4) produce localizing plotting fields on the film which show the exact

Fig. 2. The mechanical guide in position as seen posteriorly.

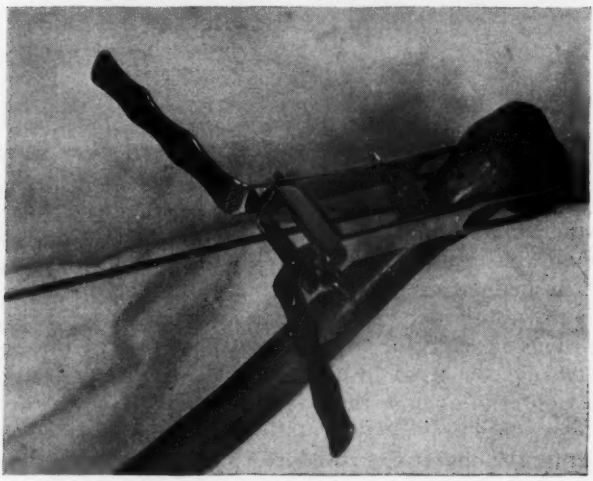

Fig. 3. The mechanical guide in position as seen laterally.

location and the right angle at which to introduce the mechanical guide. The desired line on the skin is marked with a gentian violet solution. Also before the operation the surgeon is able to determine the necessary length of the nail to be used (Engel and May).

The mechanical guide was at first used upon a cadaver. After the insertion of the guiding wire the proximal part of the femur was

removed and it was found that the wire had penetrated to the exact center of the femoral head.

Up till now the guide was used on three patients who sustained a fracture of the neck of the femur. Nitrous-oxide anesthesia was administered, although, a spinal or local novocain injection would

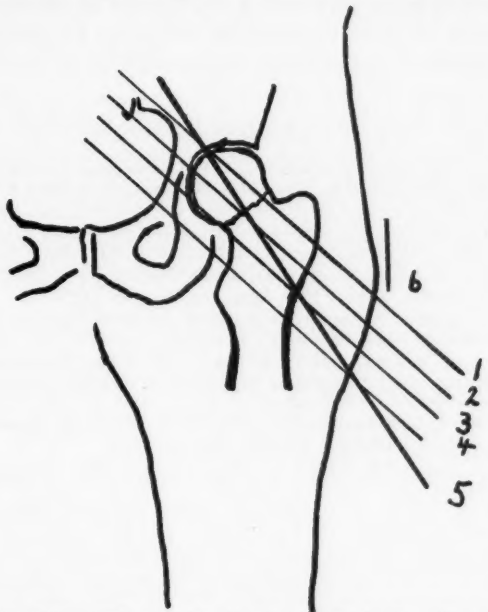

Fig. 4. Plotting fields: Wires on skin (1, 2, 3, 4). Desired gentian violet line on skin (5). 3 inch long wire on skin near the greater trochanter to determine the necessary length of the nail (6).

have been as effective. For internal fixation I used in the first case two screws, in the second four strong wires parallel to each other. In the third case a Smith-Petersen nail was directly inserted without the aid of a guiding wire. All three cases show a satisfactory position of wire, screw or nail in both views.

It should be pointed out that there is no danger connected with the placing of the mechanical guide. The prongs should be kept close to the bony surface which will give adequate protection. The femoral artery is separated from the field by the iliopsoas muscle, and the sciatic nerve lying posterior to the small outward rotators, is not in danger of being injured.

This mechanical guide is offered for the purpose of permitting a quick and accurate introduction of a wire, screw or nail without guesswork and without radiographic control. The inexperienced

surgeon should take a straight anteroposterior film on the operating table to check the position of the guide.

Up until the present time this guide has been used in only three cases and therefore it cannot be considered as adequately tested. But it seems unnecessary to defer the publication of this preliminary report because it deals only with a mechanical problem encountered at the operative procedure of internal fixation. The guide is simple, appears mechanically sound and thereby worthy of further trial.

2519 Oak Lawn

REFERENCES

1. Cleary, E. W.: An Accessory Table Top Which Eliminates Guesswork from Extra-Articular Internal Fixation of Femoral-Neck Fractures, J. Bone & Joint Surg. *20:* 449 (Apr.) 1938.
2. Engel, G. C., and May, H.: Two-Plane Direction and Range Finder for Nailing Fractures of the Neck of the Femur, J. A. M. A. *110:* 1828 (May 28) 1938.
3. Harris, R. I.: Experiences with Internal Fixation in the Fresh Fractures of the Neck of the Femur, J. Bone & Joint Surg. *20:* 114 (Jan.) 1938.
4. King, D.: A Simple Technique for Inserting the Smith-Petersen Nail, J. Bone & Joint Surg. *20:* 501 (Apr.) 1938.
5. King, D.; Leef, Edward, and Terwilliger, C. K.: The Importance of Accurate Roentgenography and Interpretation in Femoral-Neck Fractures Treated by Internal Fixation, J. Bone & Joint Surg. *22:* 168 (Jan.) 1940.
6. Myron, O. H.: Lateral Introduction of the Screw-Bolt in Intracapsular Fracture of the Hip, J. Bone & Joint Surg. *20:* 400 (Apr.) 1938.
7. Robertson, R. C.: A Leg Holder for Hip Nailing, J. Bone & Joint Surg. *20:* 782 (July) 1938.

CLASSIFICATION AND TREATMENT OF CHRONIC ARTHRITIS

K. K. Sherwood, M. D.

Seattle

THE common cold and chronic arthritis are two frequent diseases of a non-fatal character which, because of a lack of pathologic understanding, have accumulated a vast array of confusing terminology and treatments. It is the purpose of this paper to point out the certain fundamental pathologic changes occurring in chronic arthritis, to correlate these changes with clinical syndromes, and to outline what may be expected from rational therapeutic procedures. In this paper I shall attempt to place my arguments upon pathologic grounds, and emphasize the effect of treatment upon the pathology of the disease. I shall not stress the fundamental etiologic causes, but limit myself to a clinical viewpoint in dealing with causative factors.

When one analyzes the disability of arthritics, that is, stiffness and pain, one finds that the fundamental cause is swelling of the joint structures. Pathologic investigation of this swelling shows it to be of two classes; one of an inflammatory, infiltrative, proliferative character (fig. 1); the other, a purely edematous engorgement of the peri-articular structures (fig. 2). We, thus, have a pathologic foundation for the two types of arthritis, namely, the atrophic rheumatoid or infectious arthritis, and the hypertrophic osteo- arthritis or the degenerative type. One is pathologically inflammatory, the other is pathologically non-inflammatory.

If we take the atrophic or inflammatory type of joint changes and analyze the cases in which it occurs, we will find three types of patients or three characteristic syndromes. The most common is that which the English[1] most aptly call "metastatic arthritis." This is the type of arthritis which follows an acute infection, or is due to an active focus of infection. It resembles in clinical course and in distribution, gonorrheal arthritis except that it is not gonorrheal in etiology. It affects mostly the large joints of the extremities, especially the lower extremities; and there is no logical, predictable sequence of joint involvement. It tends to run a short course and to heal with the minimum of joint disability. There are only moderate constitutional symptoms, and one is impressed with the severity of the local reaction rather than the severity of the general reaction. This is most common in young adults, but may occur at any age.

The second form of atrophic or inflammatory arthritis may aptly be called peripheral atrophic arthritis. This almost always begins

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 10, 11 and 12, 1940.

gradually either in the transverse arch of the foot or in the proximal interphalangeal joints and knuckles of the hand. There is a marked constitutional reaction; the patient is obviously sick all over. His joints are not extremely painful, but stiff and swollen. They are characteristically fusiform in shape as all the joint structures are involved. If it were not for the joint complaints, the constitutional symptoms would make the diagnosis of early pulmonary tuberculosis very likely. In contrast to the severe pain suffered in the metastatic

Fig. 1A. Findings in the knee of an atrophic arthritic. Notice the thickness of the joint capsule, the inflammatory grapelike pannus and the erosion of the cartilage under the pannus.

forms of atrophic arthritis, the sufferers from peripheral arthritis do not complain so much of pain as of stiffness and swelling. The disease tends to be strikingly progressive; remissions occur only rarely except during pregnancy and jaundice; and in keeping with its very gradual onset, ankylosis is relatively early and tends to be permanent. The syndrome is so symmetrical in distribution that its grave character can be diagnosed by this symmetry. Usually, the story is not only of bilateral transverse arch involvement, but of involvement of one or more fingers on both hands. The symmetry is so exact that, as a rule, it is the same finger and the same joint involved in both hands; and the onset in both locations is usually within a month. The characteristic stories which these patients tell is that of treatment for fallen arches, then having trouble getting

their rings off their fingers, then being too tired to keep on the job any longer, accompanied by stiffness of the hands, the wrists, the feet and the ankles and occasionally the knees. It is an important diagnostic point that the spine is very rarely involved. Of equal diagnostic importance is the occurrence of rheumatic nodules. As in rheumatic fever, subcutaneous nodules are of grave importance. In atrophic arthritis they occur especially around the elbow and on the outer side of the forearm (fig. 3). They frequently become 3 or 4

Fig. 1B. Photomicrograph showing the cellular infiltration of this pannus.

cm. in diameter, are only slightly painful and freely movable. They enlarge with the activity of the disease; and, as the disease becomes quiescent, disappear. This type of disease occurs especially in women at the reproductive age, though it may occur in either sex at any age.

The third type of inflammatory arthritis is usually labeled "spondylitis." Its pathology is similar to the peripheral symmetrical variety but the distribution is strikingly different. It affects the spine, the hip and the shoulder joints; and only rarely extends to the peripheral skeleton. Clinically, we have the same marked systemic reaction which occurs in the peripheral type. The patients are obviously toxic, they are thin, have characteristically poor appetites and indefinite, atypical gastrointestinal symptoms. While their complaints may be mainly neuritic in character, they are usually accom-

panied by remarks about stiffness of the back or neck. Characteristically, the disease begins earliest in the sacroiliac joint, next in the lumbar spine, next in the cervical spine, and lastly, the thoracic spine. It occurs very rarely in women. It is a relatively severe disease and tends to be progressive without remission.

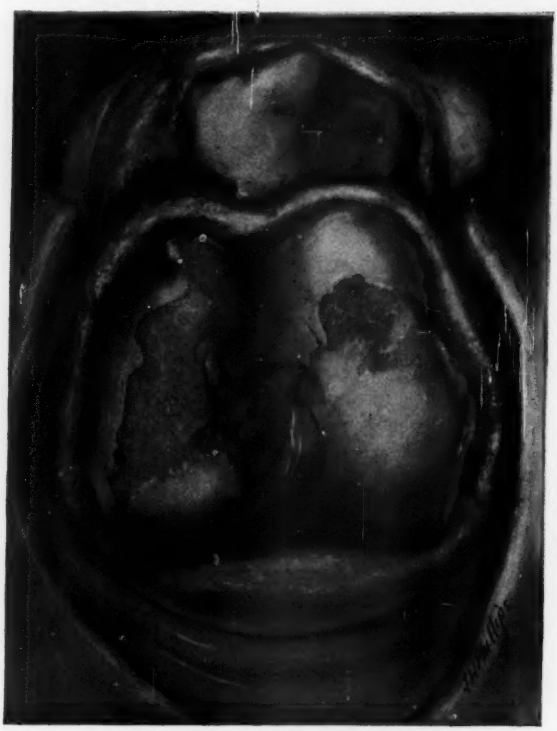

Fig. 2A. Findings in the knee of a hypertrophic arthritic. Notice the edema of the joint capsule as well as the cartilage erosion in the weight-bearing areas.

From the laboratory and x-ray standpoint, certain aids are available to help us in the diagnosis of these syndromes of inflammatory arthritis. In the metastatic arthritis we frequently have low fever, leukocytosis, moderate increase of sedimentation rate, little or no anemia and the x-ray changes are limited to the bones involved. In the peripheral type of atrophic arthritis we have a generalized decalcification of the skeleton as witnessed by the x-ray. There is a low grade temperature, a moderate to severe anemia, little leukocytosis, and extremely rapid sedimentation rate. In spondylitis, we have the characteristic fusion of the sacroiliac joint and later the calcification of the anterior longitudinal ligament of the spine. In

the laboratory, we have anemia, rapid sedimentation and no leukocytosis.

Now we come to the more common larger group of arthritides in which the cause of symptoms is non-inflammatory edema. Upon correlating this pathologic material with clinical symptoms, we find

Fig. 2B. A section of the synovia showing the edema and lack of cellular infiltration in the tissues.

that we are dealing with a number of quite different clinical syndromes. These clinical syndromes vary by having a different etiology, joint distribution and different complications. The changes within the joints are merely variations in extent and severity of the non-inflammatory edema. I wish to emphasize that changes due to wear and trauma that occur in the joint structures are merely factors favoring edema formation, and not in themselves, the cause of symptoms. The easiest syndrome to differentiate is that arthritis following trauma. Characteristically, the story is of a broken leg, a cast, stiffness, crepitation and cracking in the knee joint for one or two years. This is purely a traumatic condition, and if it had not been for the trauma, there would have been no symptoms of the disease. Undoubtedly, the symptoms are due to changes in the weight-bearing line; and usually nature, if given sufficient time, will cure this spontaneously.

The most common of this non-inflammatory group is that which occurs near the menopause in women. It characteristically affects women who are obviously suffering from hypo-ovarianism, usually with the complication of obesity. Its location is in the knee or shoulder. There may or may not be secondary symptoms in the back, the hands or the feet. The disease is usually of gradual onset and only occasionally do remissions occur. The pain is characteristically rather severe and the usual story which is emphasized by the patient is that she is stiff after sitting down for a short while. On closer questioning, we find that there has been some morning stiffness, but that the pain is usually worse in the evening after she has been on her feet all day.

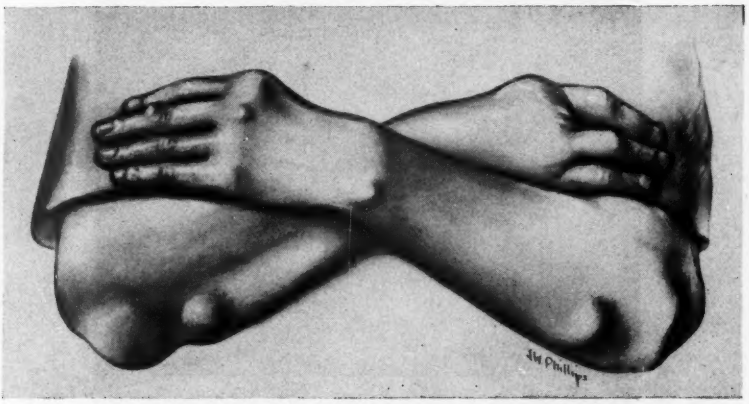

Fig. 3A. The location and size of rheumatic nodules in a case of active atrophic arthritis.

The next class has been designated morbus coxae senilis which merely means hip disease of the aged. It usually is of gradual onset, is eventually bilateral and the main complaints are loss of motion in the hip joint and pain in the anterior and inner thigh. There is no constitutional reaction; the walk is with eversion of the foot, with or without a limp due to muscular spasm. About 25 per cent of the cases date their symptoms from a serious injury.

The fourth syndrome of degenerative non-inflammatory arthritis consists of Heberden's nodes or a nodular enlargement of the terminal interphalangeal joints. Strecker[2] in Cleveland has made a statistical study of the occurrence of this disease and finds an inherited tendency to its development. Trauma, both in the form of acute trauma such as the baseball finger, and the form of chronic trauma such as seamstresses, stenographers and pianists expose their fingers to, predispose to its early development. The nodular enlargement

is partly bony, partly cartilaginous, but in the acute stage, largely edematous. There are no constitutional symptoms and the patient comes to the doctor perhaps a little shamefacedly with such a minor complaint; but it is both unsightly and awkward. It occurs, clinically at least, largely in women past 45. There is frequently more or less severe vascular unbalance.

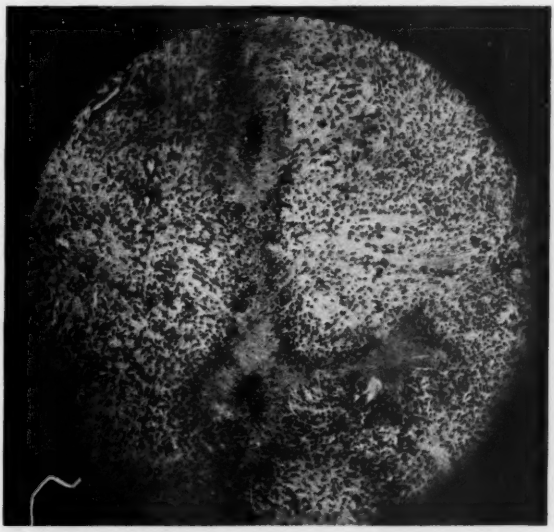

Fig. 3B. Photomicrograph showing the nodule to be made up of connective tissue with some cellular infiltration.

In addition to these more or less limited syndromes of non-inflammatory edema, there occurs a generalized form of this degenerative disease usually in men, occasionally in women in which there are multiple painful joints largely in the back and the weight-bearing areas. They are usually of acute onset, with incomplete remissions, and characteristically we find persons well past middle life trying to do the same amount of work with the same speed that they did when their joints were physiologically younger. Naturally, we have an accumulation of minor traumas with resultant edema; and with the edema comes the pain and stiffness. Almost as a special type of this form of arthritis, one may label neuritis due to hypertrophic or degenerative arthritis of the spine. Following minor trauma or more usually merely after over-use and chilling, occurs pain of root distribution not accompanied by signs of organic disease of the nerves, and the pain can be increased by pressure upon the nerves or by motion of the spine. There are no constitutional reactions and the patient is well except for the root pain. It occurs predominantly

in men, but may occur in either sex; and the patients are usually past 50 years of age.

Often coming to the doctor as self-diagnosed arthritis or neuritis are cases of displacement of the nucleus pulposus. This is a good example of a common disease complicated by a Latin name. Between the vertebral bodies there is a fibrous cartilage in the center of which lies the remnant of the embryonic notochord. This is a globular semi-fluid body. Through use and trauma the intervertebral cartilage splits and the posterior longitudinal ligament, which is weak, suffers a tear. Either a piece of the intervertebral fibrocartilage or the nucleus emerges through the tear and causes pressure upon the dural sac. This, characteristically, occurs following minor trauma. The story is that of a minor fall, back injury or sprain, heavy lifting followed by a lame back for two or three weeks; complete freedom for two or three years, a sudden wrench, a severe sciatica lasting from two to four or six weeks with gradual recovery, and from three to five years later a repetition of the same with or without complete recovery. It can occur in either sex, usually in adults past 30. Diagnostically, there are demonstrable evidences of muscle atrophy and usually motor and sensory changes present in the area which would be affected by the corresponding nerve roots. The intermittency of the symptoms is due to the fact that the acute pain is caused by swelling from motion of the displaced cartilage, this causing edema around the displacement. Pressing on the dural sac, and frequently edema of the nerve roots within it, gives rise to pain. With rest, this edema subsides and while loss of strength and occasionally slight paresthesia persist, these are usually so slight that they are overlooked by the patient. Then following minor sprains or twists, there is motion of this displaced fragment, pressing it more firmly against the dural sac, a recurrence of the edema and with this edema a recurrence of all the symptoms of the previous attack, frequently to a magnified degree.

While atrophy in the mid-thigh or the calf of the legs, absence of Achilles tendon reflex, areas of anesthesia are all characteristic of the disease; in my experience the most frequent and easily demonstrated sign is the atrophy of the muscle of the mid-thigh or mid-calf. There are no systemic signs of toxemia. Spinal puncture reveals an increased protein content of the first few cubic centimeters of spinal fluid removed which is characteristic of the syndrome. With this much to go on, lipiodol or preferably air injection of the dural sac should be performed. Either of these methods will reveal deformities of the dural sac. In the majority of cases in my experience, the severity of the symptoms does not warrant an air study of the spinal canal. Without it, a provisional diagnosis may be made and the case be treated conservatively by means of a Taylor

brace. If symptoms are not controlled, further study and operation are indicated. The results from the operation are spectacularly successful.

More interesting, but of less serious importance is spondylolisthesis. This is frequently asymptomatic, occurs largely in elderly individuals and occasionally gives rise to either local back pain or root symptoms. It is characteristically aggravated by minor motions or relaxation, as for example, sitting. I have had several patients who complained of inability to ride for long distances in the car, who upon examination showed spondylolisthesis. In at least half of the cases, a ledge can be felt on the spinous processes. When this occurs in a lumbar vertebra, and usually when occurring elsewhere, there is a history of sudden injury. The x-ray is diagnostic. The amazing thing is the mildness of the symptoms with the severity of the findings in the x-ray. Results of treatment by means of a low back brace are excellent.

No discussion of arthritis is complete without a discussion of gout. Gout is manifested as a recurring acute attack of arthritis with complete remission. Characteristically, it comes on with either a decrease in activity, increased food intake, increased alcohol intake or anesthesia. It is the most frequent form of postoperative arthritis.[3] It affects the feet, the knees, the shoulders and the elbows. The pain is the most severe of any arthritic pain, is relatively unrelieved by morphine but magically relieved by colchicum or cincophen. The blood uric acid may be elevated, but in the first or second attack, especially if seen during the recovery stage and not in the early part of the attack, the blood uric acid may be normal. The patient who gets drunk every pay day and has an arthritis the following Sunday should be suspected of having gout.

I have said a lot about the classification of arthritis. I think it is the most important part of arthritic treatment. For if one has an idea of the pathology one is dealing with, then one's therapy is self evident. As I have emphasized, the symptoms of arthritis are due to swelling. If one finds clinical evidence that the swelling is inflammatory, emphasis is placed upon measures most effective in healing chronic inflammation. If the swelling is non-inflammatory in character, one evaluates the mechanical, the circulatory and the toxic factors which may produce it.

Failure of reasonable success in the treatment of the symptoms in chronic arthritis may be classified under three heads:

1, The failure of complete diagnosis, either not finding out what the essential features of the arthritis are, or missing a complicating disease.

2, not trusting the patient; and

3, incomplete treatment.

By not trusting the patient I mean that the patient knows better than the doctor whether a medicine is helping him or hurting him. All too often we hear the story of shots given with the patient getting progressively worse, but because the shots were labeled "arthritis shots," they were persisted in, in spite of clinical evidence that they were ineffective. This I believe to be a serious and common mistake. If one particular form of the treatment is not effective in a reasonable length of time, from three to four weeks, it should be abandoned and some other form of therapy substituted. In other words, let us be sure that if we can't help the patient, we do not hurt him. I feel very strongly that when the patient comes in and tells you that he has been worse since his last treatment, surely that treatment should be repeated with the utmost caution. The third point, incomplete treatment, is, of course, the most frequent cause of failure. And what do I mean by incomplete treatment? This is what I mean: that the patient, as a whole, should be treated in all ways which will benefit him. It should be remembered that he has to live with his arthritis and the treatment should not be severe enough over any length of time to prevent him from enjoying each day as much as possible. With this in mind, treatment may be classified under nine headings.

The first and most important is rest. As I tell my patients, it is the hardest but the most important medicine to take. It should be both constitutional and local. The more acute the arthritis, the more the number of hours of bed rest. In addition, for acute pain or acute swelling, local support of the affected part especially the lower extremities by means of half shell casts, preferably made of castex, is found to be a great comfort as well as a time-saver in the reduction of swelling. The half shell cast permits removal of the affected part for massage, passive and active motion, and supplies in the interim the support needed to give muscular relaxation.

The second heading is diet. This should be individualized as practically all arthritics have some food which disagrees with them either constitutionally or with the gastrointestinal tract. As a rule, the fat patients I attempt to reduce, and the thin patients I attempt to fatten. Frequent feedings are of great benefit in both types of diet. In the subcaloric diet, feeding between meals of a small amount of fruit juice keeps the blood sugar at a reasonable level preventing weakness and irritability, while the feeding of high caloric fluids like milk and cream, will help the cadaverous individual to gain. In arthritis there is no contraindication to a high protein diet. Meats do not hurt the arthritic. Vitamins and minerals as contained in meats, vegetables, fruit and milk should be given liberally to all arthritics.

Other than rest and diet, the most important part of the treatment of chronic arthritis is the treatment of the complications. Almost all arthritics have something else wrong. The complication may not be serious or extensive but it should be remedied as part of the patient's arthritic treatment. The most common, of course, are the active foci of infection such as pyorrhea, pyelitis, prostatitis, and chronic tonsillitis. In addition, menopausal symptoms, constipation, gallbladder disease, cardiac disease and varicose veins all contribute to the continuing of arthritic symptoms. The completeness of removal of foci has varied from the early extreme of removal of all focal organs infected or otherwise to a more recent skepticism of the value of any focal removal. Now we are beginning to separate the cases. We know that in the peripheral atrophic and the spondylitic patients, removal of foci does nothing more than generally improve the patient. We know, similarly, that in the metastatic type of arthritis, the foci are the precipitating cause and their removal will frequently prevent recurrences of the disease. In the non-inflammatory type of osteo-arthritis, focal removal is largely a matter of judgment. There are distinctly three factors favoring this formation of non-inflammatory edema which gives rise to the symptoms in osteo-arthritis. First is the mechanical, second is the circulatory and third is the toxic factor. If the toxic factor is the predominant factor, then focal removal in this type of arthritis will lead to beneficial results. On the other hand, if the edema is largely mechanical or circulatory in etiology, focal removal will have little benefit.

The fourth part of the treatment of chronic arthritis is vitamins. I believe cod-liver oil, or some source of vitamins A and D is indicated in the arthritides accompanied by systemic decalcification, that is, the peripheral and spondylitic types of inflammatory arthritis. I do not believe it is indicated in the other forms of arthritis. Vitamin C as an anti-edema factor is very important in chronic arthritis. It is easily measured in the blood by a simpler procedure than blood sugar, and if low, I believe it should be replenished not only by a high fruit diet, but as a pure vitamin in from 100 to 300 mg. daily doses. I do not believe that vitamin C should be administered routinely especially in the presence of a normal sedimentation rate without its blood estimation. It is, however, harmless though expensive. Vitamin B is of much less frequent value in chronic arthritis. We have no practical quantitative measurement of its level in the blood. In general, one may say it is useful in neuritis due to chronic alcoholism, due to malnutrition or due to debility. Similarly, in root pains of toxic origin, it may give speedy symptomatic relief. Because one of the earliest signs of B deficiency is constipation, it is seldom indicated in the unconstipated individual.

Turning now to the drugs of more specific value, sulfanilamide and allied compounds should first be considered. In general, these are ineffective except in the metastatic type of arthritis or occasionally in a rapid progressive form of the peripheral type of atrophic arthritis. They are best combined with the next form of specific therapy, namely fever therapy.

Fever therapy may be given by means of a hot air cabinet, by electrical induction or by the injection of foreign protein. I believe that, in non-specific chronic arthritis of the metastatic type, perhaps typhoid administration is the method of choice. What we wish to procure by the use of the fever is a marked systemic shock manifested by an increased leukocytosis and increased activity of the reticulo-endothelium. This is what typhoid does and to some extent fever therapy. However, those of you who have given both forms of therapy, I am sure, will feel that the typhoid is much the less severe.

The next type of special therapy to be considered is vaccine therapy. Vaccines may be autogenous or stock. I believe the stock vaccines are superior to the autogenous because the concentration is constant and measurable. It is important in administering vaccine, whether it be given intravenously or subcutaneously, to appreciate that optimal dosage is followed by forty-eight or more hours of subjective improvement. This sensation is accompanied by a slight leukocytosis.[4] If too large a dose is given, the patient will be markedly worse, and if too small a dose is given, there will be no effect.

A lot has been written in recent years upon the value of gold in atrophic inflammatory arthritis. Probably in the peripheral atrophic inflammatory type of arthritis, it is the drug that should be first used. For five years I have used the gold sodium thiosulphate salt and can testify that it is effective though dangerous. I have used consistently a very small dose, 10 mg., repeated not oftener than twice a week. In either the very acute cases, manifested by high leukocyte count, or the chronic relatively quiescent cases manifested by a normal sedimentation rate, I have found it to be symptomatically ineffective. When gold is administered, before each dose the patient should be questioned in regard to the three signs of gold toxicity: pruritus, purpura and hematuria. The pruritus is usually harmless if the gold is stopped when it first develops; if not, there follows an exfoliative dermatitis which may persist for months. The purpura is due to thrombocytopenia, and if the gold is stopped promptly, I have found intramuscular liver and vitamin C given daily will control the platelet deficiency in a period of days. Hematuria is the rarest form of gold toxicity. I have recognized four

cases in five years, three in spondylitics, one of which had to be transfused to control the condition.

Theelin has a definite role in the treatment of arthritis. Recent work has shown that it is a vasodilator and increases the volume of blood in an extremity. This is in keeping with the clinical evidence that in Heberden's nodes it is frequently of value in the control of local pain and swelling. In the degenerative forms of arthritis, when I feel the edema is partly a matter of circulatory stasis in contrast to either varicose veins or cardiac decompensation, I have found theelin to be a very effective drug.

General tonics are occasionally of use, arsenic being the best. In the last six months I have routinely used intramuscular vitamin C with every injection of intravenous gold as a means of preventing toxicity.

BIBLIOGRAPHY

1. Ray, M. B.: Rheumatism in General Practice, page 83. London: H. K. Lewis & Co., 1934.
2. Stecker, R. M.: Exhibit, American Medical Association, St. Louis, May, 1939.
3. Hench, P. S.: Comments on the Diagnosis and Management of Gout in Certain Parts of the United States, Proc. Staff Meet., Mayo Clin. *12:* 262-269, 1937.
4. Unpublished work of the author.

AMEBIASIS: A CAUSE OF ABDOMINAL PAIN

FRANCIS M. MASSIE, M. D.
Lexington Clinic
Lexington, Kentucky

THE words of Sir Thomas Vicary in 1577[1] are so appropriate that this discussion is opened with them: "Every chirurgion ought to be learned and [in] that he knows his principles, not only in chirurgie but also in phisicke that he may better defend his chirurgie." In Sir Thomas' time too, surgeons had to be on the defense, especially lest they mistreat by surgery those patients who should have been left to the physician.

It may be a healthy sign that a group of surgeons find themselves addressed by another surgeon on a so-called "medical" subject. It is granted readily that this could be better done by a physician. But I like to think that "the message" comes with peculiar force from one who has already fallen into the very errors he would avoid in the future.

So much has been said, quite properly, on what conditions require surgery and how this surgery should be done that it may be worth while to turn our attention briefly to a disease which demands that we "withhold the eager hand." When with knife and hemostat we blunder into an abdomen already occupied in part by Endamoeba histolytica, punishment is quickly visited on us and our patient. The mortality following surgery in the acute fulminating type of disease is shockingly high.[2, 3] Failure to recognize the true condition as amebiasis is usually because we fail to think of amebiasis at all.

It is my purpose to present to you a few practical points in the diagnosis of amebiasis and because pain is the symptom which misleads us most often I have chosen to emphasize its importance by placing it in the subtitle. In this article will be no discussion of the literature nor history of the disease, nor more than mere reference to its incidence, laboratory diagnosis and treatment.

It is unfortunate that for many years the term "amebic dysentery" has been so widely used. As Mackie[4] says, "Conventional terminology has assigned to the infection the name of one of its less common clinical manifestations." The medical profession is beginning to realize that dysentery is merely a symptom which may be an important index to the severity of the disease in the individual, but has no vital relation to the presence or absence of the Endamoeba histolytica. Amebiasis, a term which is rapidly supplanting amebic dysentery, means infestation of the human intestine by the protozoon Endamoeba histolytica. We know that this infestation may cause

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

little or no disturbance in the host, though Craig[5] thinks that the majority of "symptomless" carriers suffer from a chronic toxemia due to the often unrecognized infection.

Another advance in our conception of amebiasis lies in the growing knowledge that it is not a tropical disease. That the first case ever reported occurred in St. Petersburg, Russia,[6] in a person who had never been in the tropics, escaped the attention of the profession. Of course, for many years there have been scores of men thoroughly familiar with the incidence and distribution of amebiasis but that it quite probably exists in one of every five to twenty[5,8,9,10,11,12] persons we meet in our daily business and social contacts has not been generally understood by doctors throughout this country.

Often analyses do not give accurate pictures of the real incidence. For example, our own study of fifty-two cases occurring in private practice in the Lexington Clinic from 1934 to 1939, shows that these were from a total of slightly more than 15,000 admissions, an incidence much lower than the true one. On the other hand of the last 69 persons sent to the laboratory for "ameba hunts," 14 have been reported positive. This figure, 20 per cent, is undoubtedly higher than that for our population in central Kentucky as a whole. The true incidence is reached only from studies made on cross sections of city and rural populations of both healthy and sick. Many such careful studies have been made which show the incidence to be somewhere between 2 and 30 per cent with most figures lying between 5 and 20 per cent.

In making a clinical classification most observers have followed that of Craig[11] who places infected persons in four classes:

1. Those with no symptoms, the so-called "carriers." Many of these on close questioning will be found to have symptoms which disappear after specific treatment.

2. Those who have indefinite symptoms mostly referable to the gastrointestinal tract and nervous system. Vague abdominal pain, anorexia, nausea, pain during or soon after eating, headache, gas, weakness and general malaise are prevalent complaints; in short, "the neurasthenic."

3. Those with recurrent diarrhea with or without passage of blood or mucus, with periods of comparatively good health between attacks.

4. The acute or chronic amebic dysentery, with prostration, fever, or both, often with ulcerative colitis. In this class should be placed the acute fulminating type of disease seen in the Chicago outbreak in 1933. To this we would add a fifth group, those with symptoms unrelieved by specific treatment and antispasmodics. These obviously suffer from concurrent ailments which may be masked by finding the pathogenic ameba in the stools.

In our small series of 52 patients table 1 shows the age distribution: 38 per cent between 25 and 35 years and another 17 per cent between 35 and 45. This corresponds with the experience of others.

TABLE 1

AGE DISTRIBUTION OF CASES OBSERVED

Age Groups	*Total*	*Per Cent of Total*
16-20	5	9.62
21-24	5	9.62
25-29	12	23.08
30-34	8	15.38
35-39	3	5.76
40-44	6	11.53
45-49	3	5.76
50-54	1	1.93
55-59	2	3.85
60-64	4	7.69
65 Plus	1	1.93
Unknown	2	3.85
Total	52	100.00

Morledge[12] found in 532 persons studied by the Department of Parasitology of the University of Oklahoma that the incidence was 8 per cent and that the greatest incidence occurred between 40 and 50 years of age. He quotes Craig as finding the peak between 26 and 30 with a sharp decline after the age of 35. We found 28 females and 24 males as contrasted with Morledge's 80 per cent of positives in men. The preponderance of females in our group is possibly explained by the fact that all of ours had symptoms and consulted a doctor. Women are more likely than men to do this for what they consider minor disturbances.

We have not been able to study in our group the source of infection, chiefly because most of our cases fall into classes 2 and 3. Sources cannot be traced except in the epidemic type of disease. Almost all observers agree that the endemic variety of infections, because it has to be swallowed to gain entrance in man, is carried from one person to another by raw food, fingers and probably by flies. The epidemic type is almost certainly due to gross contamination of the drinking water. We know that the vegetative form of the ameba, the active pathogenic organism, is quickly killed by the juices of the stomach and intestine.[6] It is the cystic form which when swallowed survives the passage through the stomach and greater part of the small intestine and in the terminal ileum and colon is transformed into a four nucleated ameba which divides into eight small amebae.[6] These are the active pathogenic organisms, and as the name Endamoeba histolytica implies, burrow into the mucosa and dissolve the cells immediately around them. They then form ulcers which become secondarily infected by bacteria, or they may burrow for long distances causing necrosis in the overlying

mucosa and the typical ulcers with overhanging edges of normal mucosa, or they may be indolent and produce no gross lesions.

Brown and Magath[13] think that the importance of "carriers" and raw food in the transmission of the disease has been overestimated largely by reason of the work of Spector and Buky[14] who showed that the cysts are not viable on the hands for more than ten minutes. Most workers in the field agree with Craig[6] that this is long enough to transmit the disease from one individual to another.

TABLE 2

SUBJECTIVE SYMPTOMS RELATIVE TO AMEBIASIS ON ADMISSION

Items Considered	*Patients*	*Per Cent of Total*
Abdominal pain	49	94.23
Diarrhea (intermittent)	25	48 07
Bloody stools	16	30.76
Mucus in stools	12	23.07
Flatulence	30	57.69
Vomiting	4	7.69
Malaise	13	25.00
Loss of weight	13	25.00
Constipation	17	22.69
Normal bowel action	9	17.30

This summary indicates that the most constant subjective symptoms are in order of their frequency as follows: Abdominal discomfort or pain, flatulence, intermittent diarrhea, constipation, bloody stools, malaise, loss of weight, mucus in the stools, apparently normal bowel movements and malaise. This study would indicate that abdominal discomfort or pain and flatulence is enough to be suspicious of amebiasis infection. The type of diarrhea experienced by most of these patients was of an intermittent nature.

Table 2 shows that abdominal pain is a fairly constant symptom in those presenting themselves for study. Ninety-four per cent of our patients had abdominal pain of some sort, while less than half (48 per cent) had *ever had any diarrhea.* The pain was, in some, constant and aching, radiating at times down the legs, not sharp or lancinating in the legs but "a vague, dead ache." In some the pain was colicky occasioned or increased by eating and often in the right lower quarter of the abdomen. In many there was local tenderness over "the appendix area."

Sixteen of our patients had had no definite diagnosis before we saw them. The other 36 had been treated for many things; most prominent in the list of diseases were appendicitis, mesenteric lymphadenitis, ovarian cyst, chronic salpingitis, endometriosis, bacillary dysentery and chronic ulcerative colitis.

Table 3 is of interest in showing the time interval between onset of symptoms and correct diagnosis.

TABLE 3

THE LENGTH OF TIME BETWEEN ONSET OF SYMPTOMS AND RECOGNITION OF AMEBIASIS AT LEXINGTON CLINIC

Interval of Time	*Total Cases*	*Per Cent of Total*
Less than 1 month	5	9.62
1-4 months	5	9.62
5-9 months	3	5.76
10 mos.-1 year	0	0.00
1- 4 years	24	46.16
5-10 years	9	17.30
10 years and over	6	11.53
Total	52	99.99

This study shows that 39 of these patients had suffered from 1 to more than 10 years before admission to the clinic. The majority of these patients had been seen by physicians and many of them operated on by members of the surgical profession.

TABLE 4

OPERATIONS BEFORE DIAGNOSIS OF AMEBIASIS WAS MADE

Items Considered	*Number Patients*	*Per Cent of Total*
Chronic appendicitis	4	7.69
Ovarian cysts	2	3.85
Tonsils and adenoids	7	13.46
Fibroids	1	1.92
Kidney	1	1.92
Hernia	1	1.92
Others	2	3.85
Total	18	34.61

Table 4 is perhaps of greatest interest to us as surgeons because it shows that 34 per cent had had operations of some sort before the correct diagnosis was made. In two of the tonsil cases, one fibroid and one hernia, these operations were certainly necessary; the other fourteen patients had what we believe was unnecessary surgery. Fortunately for these patients who had abdominal surgery in no case was the condition one of acute fulminating amebiasis and no deaths resulted. In our short series there was only one death in a patient correctly diagnosed as chronic ulcerative colitis and peritonitis, proved at autopsy to be amebic colitis previously unsuspected. Does this low mortality mean difference in strains of virulence in the ameba or difference in specific resistance factors in the host? Probably neither. Craig[15] has shown that specific antibodies

disappear rapidly when the organism is eradicated and thinks clinical variations are due to the number of cysts ingested and to the general resistance and state of health of the individual. For these reasons the manifestations are usually more severe in the tropics than in temperate climates. The fact that there were no "daughter epidemics" following the Chicago outbreak confirms the opinion that the virulence in this instance was not due to any unusual strain of ameba but to overwhelming numbers of cysts ingested.

In a condition where the symptoms are so varied the diagnosis can never be made on history and physical findings. The sole criterion is the demonstrable presence of the Endamoeba histolytica in the stools. Search for the ameba should be made only by those specially trained in this laboratory procedure. As there are many protozoa in the stools of man, some of them quite similar to the histolytica and none of them pathogenic in the same sense, a report by the occasional searcher is totally unreliable. Our technic is to have the patient come to the laboratory at 9 a. m. with no more than a cup of coffee or fruit juice for breakfast. There he is given two ounces of magnesium sulphate. Receptacles are kept warm in the laboratory and liquid stools examined *as soon as passed.* Three such stools in the same morning are examined before a negative report is given. If the report is negative, a second trial a week or more later may be undertaken. In no case have we found the ameba in the later search where it could not be found in the first three stools. Magath[17] finds in 60,000 examinations at the Mayo Clinic that of those who harbor this protozoon 80 per cent are found in the first saline stool, 15 per cent in the second and 5 per cent in the third. Other observers[3, 10, 18] think examination by staining methods of the solid stool specimen gives a higher per cent of positive readings. In this method the ameba is in the cyst stage and is quite characteristic on staining. The latter method has the great advantage of not requiring the presence of the patient and the convenience of examining the specimen at any time before it becomes dry. Obviously examination of large groups must be by this method. Patients who are passing bloody mucus do not require the saline cathartic though even in these the salt brings out the organisms in greater numbers. The criteria for diagnosis of the vegetative or active pathogenic ameba in the saline stool are active motility, clear glass-like pseudopods, and ingested red blood cells. Magath[17] says, "for practical purposes one may, with assurance, call any ameba containing erythrocytes Endamoeba histolytica."

We have been treating our cases until recently by the following plan: 1 grain of emetine hydrochloride is given by hypodermic daily for six days. This is followed by one capsule of carbarsone (4 grains) by mouth two or three times a day for two weeks, some-

times three. Three weeks from the beginning of the treatment the emetine and carbarsone are repeated. The stool is examined and if negative no treatment is given for six or eight weeks. If symptoms continue or recur, antispasmodics are used and if these are relieved, no further treatment is given but stools are examined every six months. Treatment is repeated if symptoms recur or organisms are found in the stools. Patients who do not tolerate arsenicals we have treated with chiniofon, 3 pills three times a day (i. e. 12 grains a day) for three to four weeks.

Recently we have stopped using emetine except in those patients with bloody diarrhea and tenesmus where it is quite effective in relieving symptoms. There is evidence that emetine alone is not a reliable drug.[19, 20]

Table 5 gives the results of treatment as obtained so far.

TABLE 5

THE RESULTS EMETINE HYDROCHLORIDE AND CARBARSONE TREATMENT

Items Considered	*Number Patients*	*Per Cent of Total*
Complete relief	21	40.38
Improved	20	38.46
Unimproved	1	1.92
Unknown	10	19.23
Total	52	99.99

In the unknown group 9 of these patients are still under observation, since their period of treatment was initiated in the latter part of 1939, and one died before treatment was initiated. The twenty patients who were improved in all probability had ulcerative lesions that require time to heal.

It is important to realize that there is no quick cure for amebiasis[16]; that emetine is a dangerous drug since its effective therapeutic range overlaps its toxic range[20]; that there is no single drug which will cure all cases,[9] but that by and large the several drugs are sure to bring relief in more than 90 per cent of cases. This is a high level of efficiency in the treatment of any disease. For the relief of symptoms that persist after stools are negative, we have used kaolin and bismuth, occasionally atropine, and more recently trasentin which is highly regarded by some as an antispasmodic. Of all the drugs carbarsone introduced by Reed[20] and his co-workers in 1932 is the most valuable.

As table 6 shows it is important that amebiasis should not be allowed to mask other conditions which require surgery.

Others have made the same observation.[16, 21] Nearly 8 per cent of our patients required subsequent surgery for relief of their symp-

toms; two were relieved by removal of the appendix, one had small fibromas in the uterus, and one had endometriosis.

TABLE 6

OPERATIONS AFTER DIAGNOSIS OF AMEBIASIS WAS MADE

Items Considered	*Number Patients*	*Per Cent of Total*
Hysterectomy	2	3.84
Appendicitis	2	3.84
Total	4	7.68

The problem of amebiasis should engage our attention as surgeons because it is present in from six to fifteen million of our people. In half of these at least there are symptoms and the symptoms are primarily those of so-called neurasthenia and of various conditions which demand surgery. The only way in which we can be sure of the diagnosis is by examining the stools. This should be done only by those experienced in telling the pathogenic ameba from the several others. This paper may be construed as a plea to examine the stools of all patients coming to us where the diagnosis is not clear but suggestive of neurasthenia or of "surgical" conditions. The former group are often dismissed as unworthy of our attention and the latter meet the more disagreeable and hazardous fate of unnecessary surgery.

REFERENCES

1. Gatewood: Surgery in Amebiasis, Illinois M. J. *71:* 41-45 (Jan.) 1937.
2. Hogan, E. P.: Endamoeba Histolytica. Incorrect Diagnosis and Treatment a Potential Surgical Catastrophe, Am. J. Surg. *28:* 498-530 (May) 1935.
3. McCoy, G. W., and Hardy, A. V.: The Clinical Diagnosis of Amebic Dysentery, J. A. M. A. *107:* 1357-1359 (Oct. 24) 1936.
4. Mackie, T. T.: The Diagnosis and Treatment of Intestinal Amebiasis, New York State J. Med. *35:* 261-266 (March 15) 1935.
5. Craig, C. F.: Symptomatology, Diagnosis and Treatment of Carriers of Endamoeba Histolytica, J. A. M. A. *90:* 1345-1349 (Apr. 28) 1928.
6. Craig, C. F: The Epidemiology of Amebiasis, J. A. M. A. *103:* 1061-1063 (Oct. 6) 1934.
7. Munson, S. E.: The Clinical Aspects of Amebiasis, Illinois M. J. *71:* 21-25 (Jan.) 1937.
8. Lynch, K. M.: Prolonged Influences and Complications of Intestinal Amebiasis, J. A. M. A. *103:* 1147-1150 (Oct. 13) 1934.
9. Mackie, T. T.: Discussion Diagnosis and Treatment of Amebiasis, by Simon, S. K., Am. J. Digest. Dis. & Nutrition *1:* 486-488 (Sept.) 1934.
10. Nauss, R. W., and Salinger, M. H.: Amebic and Other Intestinal Protozoal Infestations in Representative Groups of New York City, Am. J. Pub. Health *25:* 819-828 (July) 1935.
11. Craig, C. F.: The Diagnosis and Treatment of Intestinal Amebiasis, South. M. J. *25:* 1207-1212 (Dec.) 1932
12. Morledge, W.: The Problem of Amebiasis as Seen in Oklahoma, South. M. J. *30:* 214-217 (Feb.) 1937.

13. Brown, P. W., and Magath, T. B.: Amebiasis: Diagnosis, Prevention and Treatment, Minnesota Med. *18:* 515-521 (Aug.) 1935.

14. Spector, B. K., and Buky, F.: Viability of Endamoeba Histolytica and Endamoeba Coli; Effect of Drying, Pub. Health Rep. *49:* 379-385 (March 23) 1934.

15. Craig, C. F.: Further Observations Upon the Complement Fixation Test in the Diagnosis of Amebiasis, J. Lab. & Clin. Med. *18:* 873-881 (June) 1933.

16. Tenney, A. C.: The Present Concept of Endamoeba Histolytica Infestation, Illinois M. J. *70:* 145-148 (Aug.) 1936.

17. Magath, T. B.: The Laboratory Diagnosis of Amebiasis, J. A. M. A. *103:* 1218-1224 (Oct. 20) 1934.

18. Wenrich, D. H.; Stabler, R. M., and Arnett, J. H.: Endamoeba Histolytica and Other Intestinal Protozoa in 1,060 College Freshmen, Am. J. Trop. Med. *15:* 331-345 (May) 1935.

19. Brown, P. W.: Results and Dangers in the Treatment of Amebiasis. A Summary of Fifteen Years' Experience at the Mayo Clinic, J. A. M. A. *105:* 1319-1326 (Nov. 17) 1935.

20. Reed, A. C.: The Treatment of Amebiasis, J. A. M. A. *103:* 1224-1228 (Oct. 20) 1934.

21. Paulson, M., and Andrews, J.: The Role of Symptoms and Signs in Amebiasis, Ann. Int. Med. *13:* 64-67 (July) 1939.

GASTRO-JEJUNAL ULCERATION

JAMES W. GIBBON, M. D., F. A. C. S.
Charlotte, N. C.

GASTRO-JEJUNAL ulceration is an established clinical entity, and occurs almost solely as a complication of operation for peptic ulcer of the stomach and duodenum. Since the first description there has been reported in the literature a progressive increase in its incidence, so that today one is not only concerned with the efficacy of the surgical treatment of the primary ulcer, but almost equal consideration must be given to the prevention of gastro-jejunal ulcer as a complication. While there is still a wide difference of surgical opinion regarding many phases of this secondary ulcer, nevertheless a great deal of useful and helpful information regarding its prevention, recognition and treatment has accumulated.

CAUSATION

A. Acid Values. In considering the causation of gastro-jejunal ulcer the use of clamps and unabsorbable suture materials have been discarded. Absorbable sutures have not abolished jejunal ulcer. The vulnerability of the jejunum to the acid gastric juice is considered the one most potent factor in the production of secondary ulcer. The Mann-Williamson operation in the experimental animal has demonstrated beyond question that the production of ulceration in the jejunum can be accomplished almost at will by subjecting the mucosa of the jejunum to direct contact with the acid gastric contents without the neutralizing effect of the biliary and duodenal secretions. Thus, the value of any surgical procedure in the treatment of peptic ulcer, and the degree of prevention of jejunal ulcer, must depend largely upon the extent to which the operation reduces, dilutes or completely eliminates the gastric acidity. Moynihan and Judd maintain that persistent hyperacidity following an operation is the most important single cause of postoperative jejunal ulcer. Also it has been shown that the lower the site in the jejunum exposed, the greater grows the vulnerability to the acid. This might be significant in the use of so-called long loop anastomoses between the stomach and jejunum, chiefly exemplified by the anterior gastro-enterostomy and antecolic Polya type of gastrectomy. These gastro-jejunal anastomoses by utilizing lower sites in the jejunum than the short loop, usually postcolic anastomoses, theoretically might be expected to harbor a greater hazard of secondary marginal ulceration. Statistics by various investigators indicate that such is not the case, particularly if the anastomosis is so made that the

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

efferent or distal jejunal loop is attached at the greater curvature so that as stated by Kelly[12] "the alkaline juices from the duodenum lave the stoma in its whole extent from above downward and makes alkalinization more diffuse than if the small bowel is fixed to the stomach in the opposite way, that is, isoperistaltically."

In long loop anastomoses, typified by the anterior gastro-jejunostomy and antecolic Polya resection, frequent use has been made of an entero-anastomosis between the proximal and distal loops in order properly to drain the long afferent loop. Without such anastomosis, this loop may find some difficulty in discharging its contents, resulting in a vicious circle. Some operators, however, have abandoned its use as unnecessary, and even undesirable, chiefly for the reason that such a procedure may drain away the alkaline secretions of the proximal loop from the suture line where they are needed as protective agents against the acid gastric secretions. Walton records a 7.06 per cent incidence of gastro-jejunal ulcer in 85 cases of duodenal ulcer treated by anterior gastroenterostomy without an entero-anastomosis. In contrast, he reports 25 cases of duodenal ulcer treated by an anterior gastroenterostomy with an added entero-enterostomy showing a gastro-jejunal ulcer incidence of 24 per cent. In both series the secondary ulcerations were operatively proven. If the entero-anastomosis is utilized in long loop anastomoses, the opening between the jejunal loops should be quite small, only large enough to prevent distention, and not so large as to drain away all of the alkaline secretions.

B. Indications for Operation in Primary Ulcer. Ill-advised operations for primary peptic ulcer may be a prevalent cause of secondary jejunal ulceration. It has been well said that "the more definite the indications for operation in peptic ulcer, the less likelihood of postoperative jejunal ulcer."[8] The consensus is that no surgery at all should be undertaken until there has been a prolonged and vigorous effort at relief by medical procedure. Intractability, hemorrhage, perforation and obstruction are the usual indications for surgery.

An error in the choice of the operative procedure may be the causative factor in jejunal ulcer formation. A considerable proportion of surgical opinion agrees that a gastroenterostomy done for marked stenosis of the duodenum with a low gastric acidity and motor insufficiency, gives results as good as any in gastric surgery. Under such conditions the fear of jejunal ulcer may be minimized. Eggers[6] states that most failures of gastroenterostomies are due to the performance without proper indication. Steten states "that postoperative ulcer after gastroenterostomy for cicatricial stenosis is in our experience rare."

The location of the primary ulcer should have much to do with the choice of the operative procedure. Gastro-jejunal ulcer is so much more frequently the result of the surgical treatment of duodenal ulcer than gastric ulcer, that it may well be considered as essentially a complication of the surgery for duodenal ulcer. Except in the cases of cicatricial stenosis, as above referred to, some other type of operation than the gastroenterostomy may be necessary in duodenal ulcer if marginal ulcer is to be prevented. "To those surgeons who have abandoned gastroenterostomy for duodenal ulcer, or who employ the procedure with misgiving because of the variable frequency of marginal ulcer" Hunt[10] recommends "the use of a lateral type of gastroduodenostomy." By utilizing the "subpapillary," more intensely alkaline portion of the duodenum in the anastomosis, a greater degree of neutralization or dilution of the gastric acids is obtained, with a low incidence of secondary ulceration, and the further advantages of conservatism and a low operative hazard. Reinhoff[17] has recently suggested in certain cases of duodenal ulcer, the resection of the pyloric antrum ("minimal resection") with a short loop antecolic gastro-jejunostomy. The antrum, the "pace-maker" of the stomach, produces gastrin, a hormone body similar to histamine which stimulates the acid production of the rest of the stomach as well as the motor activity. Resection of the antrum "combines the attributes of simplicity and safety as well as the restoration of the production of gastric secretion to approximately normal." While probably not enough time has elapsed since the employment of the operation, there have been to date no recurrences or secondary ulcerations following its use.

Pyloroplasty in the treatment of duodenal ulcer has been abandoned by many surgeons. Pyloroplasties, unless use is made of more extended incisions into the duodenum and stomach, have incurred the criticisms that they utilize a rather weakly alkaline segment of the duodenum (the suprapapillary, or that portion above the bile and pancreatic secretions), are too frequently followed by a second ulceration in the line of anastomosis, and have a limited operative application.

In gastric ulcer, gastroenterostomy may be employed with good results and without the same degree of risk of jejunal ulcer as encountered in duodenal ulcer. Duodenal ulcer is more regularly attended by high degrees of acidity than gastric ulcer. Hurst and Stewart[11] found that 61 per cent of duodenal ulcers were accompanied by hyperacidity, while only 29 per cent of gastric ulcers showed increased acidity.

Radical gastrectomy may often be the operation of choice for duodenal or gastric ulcer, when gastroenterostomy is not applicable.

C. Diet. Probably some surgeons make the mistake of not insisting on a postoperative dietary regime to prevent the complication of jejunal ulcer. Wilkie regards "the operation not as a cure but rather as an incident in the treatment of peptic ulcer, which should be followed by a careful medical regime. Failure to do this causes the complication of jejunal ulcer." He states "that the sensitive jejunum should be sheltered from the hyperacid gastric juice until such a time as it has acquired immunity." Lahey[14] states that "there has very definitely been an improper tendency to let patients assume that one of the rewards of a successful operation for peptic ulcer is that they may escape the rather irksome necessity for a permanent modification of eating, drinking, smoking and living habits."

D. Ulcer Diathesis. A peculiar and unusual degree of tissue susceptibility to ulceration, sometimes called "ulcer diathesis," seems manifest in some patients, and is then responsible for postoperative jejunal ulceration. It is generally agreed that no form of conservative surgery will control the ulcer forming tendencies inherent in these individuals, and that primary radical gastrectomy should be applied if they are to be freed from the hazard of recurrence. Since there is no scientific way of discovering this tissue susceptibility, the recognition of these patients presents a serious problem.

E. Contributing Factors. The excessive use of alcohol or tobacco, or the failure to eliminate all foci of infection in the teeth, tonsils, gallbladder or appendix, may be considered the inciting cause of gastro-jejunal ulcer. Sex plays a part in that men are more frequently affected than women. The Anglo-Saxon is thought to be less predisposed to ulceration than the Jew or German.

Incidence

The incidence of postoperative gastro-jejunal ulceration is a matter of considerable difference of surgical opinion. Advocates of conservative surgery state that gastric ulcer is rarely complicated by gastro-jejunal ulcer, whether treated by gastroenterostomy or other surgical procedures. Their incidence after gastroenterostomy for duodenal ulcer is from 2.8 per cent to 5 per cent (Balfour[1, 2] 3.2 per cent; Heuer[9] 3.5 per cent; Luff[15] 2.8 per cent; Walton[20] 3 to 4 per cent; Kelly[12] 2 to 5 per cent). They thus maintain that radical gastrectomy is unnecessary in the vast majority of patients to cure the ulcer, adds a needless sacrifice of the healthy portion of the stomach and at a greater surgical risk. Radical

surgery, it is believed, should be restricted to a relatively small group of patients in whom a high degree of tissue susceptibility might be suspected.

Advocates of radical gastrectomy maintain that the gastro-enterostomy should be abandoned as a method of treatment since it fails to reduce the acidity of the gastric secretions and is followed by gastro-jejunal ulceration in 15 to 34 per cent of cases (Berg,[3, 4] Lewisohn, Strauss, Lahey,[14] Haberer, Finsterer,[7] Ogilvie[16]). In all cases of gastric or duodenal ulcer which have resisted medical treatment, resection of two-thirds to three-fourths of the stomach is considered to be the only operative procedure that will produce anacidity and so safeguard the patient against another ulcer. Increasing experience however has shown that radical gastrectomy does not invariably control the gastric acidity and that secondary ulceration occurs in a definite percentage of the patients. These investigators nevertheless maintain that while gastrectomy may not give complete immunity to the onset of marginal ulcer, if enough stomach is removed to produce achlorhydria the complication is unknown.

Diagnosis

The diagnosis of gastro-jejunal ulcer offers no serious difficulty as a rule. The patient who has had a previous operation for peptic ulcer, after a period of variable relief, returns complaining of symptoms highly significant of ulcer, although these symptoms may lack the regularity, periodicity and control manifested in the primary ulcer. The pain is more commonly located to the left of the midline, in or near the upper abdominal quadrant. Vomiting may occur but may not be marked unless fibrosis and stenosis exist in the lesion. Gross hemorrhage occurs in a large percentage of the cases and indeed a sudden and profuse hemorrhage may be the first and only sign of the ulcer. With the progress of the lesion and the development of complications, the symptoms are greatly augmented. Penetration and attachment to the peritoneum of the abdominal wall in jejunal ulcer following an anterior gastroenterostomy intensifies the pain to an unbearable degree, with development of localized tenderness, rigidity and even a mass. Fixation and rupture of the ulcer into the transverse colon with the development of a gastro-jejuno-colic fistula after a posterior gastroenterostomy produces profound disturbances manifested by an intense diarrhea on taking of food of any kind, marked loss of weight and strength, the belching of flatus, rarely fecal vomiting, dehydration and anemia.

X-ray films may reveal the ulcer crater if there is depth and penetration, but a superficial ulcer may be obscured by the usual

irregularities which are seen around the gastroenteric stoma. A barium enema is often necessary before x-ray visualization of a gastro-jejuno-colic fistula can be obtained. The barium can then be seen passing from the transverse colon into the stomach. The gastroscopé furnishes a further means of diagnosis in doubtful cases.

Treatment

There seems to be no reason why a medical regime should not be given a trial in cases of gastro-jejunal ulcer. Medical treatment, if successful at all, is never so effective as it is in the case of the primary ulcer. The jejunal lesion manifests a far more progressive habit than the usual peptic ulcer, probably because the control is more uncertain and difficult. This should, however, be strictly confined to the early cases in which the symptoms are mild or of recent development and the crater is small or inconspicuous. But with a repetition of hemorrhage, the persistence of pain or intractability and penetration, surgical treatment should be promptly advised. Furthermore, since the complications of jejunal ulceration are so grave and the surgical treatment so hazardous when they exist, that it would seem unwise to prolong unnecessarily a medical plan which is not proving effective. Some authorities are even of the opinion that surgery is usually indicated after the diagnosis is made, believing that any medical treatment is of little or no value in healing the lesion.

Theoretically, if the pylorus and first portion of the duodenum are patent and the original ulcer healed, disconnection of the gastroenterostomy should be sufficient to cure the secondary ulcer. However, in actual practice it is not sufficient, chiefly because it neglects to deal with at least two important features present in the patient with recurrent ulcer. The first is, as stated by Walters, "that the patient with jejunal ulcer has a definite predisposition to the development of ulceration or a tissue susceptibility to ulceration, and second, that the ulcer forming tendencies of that individual have not been adequately controlled by the procedure that has been performed." To stop with simply a disconnection of the gastroenterostomy and excision of the ulcer leaves these factors unaltered and experience has shown that in many cases it is only a question of time before there is recurrence of the old ulcer, or the formation of a new one in the stomach or duodenum.

Therefore, radical gastrectomy of the Polya or Hofmeister type must be added in order to alter profoundly the gastric physiology and to control permanently the gastric acidity. Furthermore if complete anacidity is to be produced by the resection, all of

the pylorus should be included as suggested by the work of Edkins. Ogilvie noted that the cases in which he did not include the pylorus had a higher gastric acidity after gastrectomy, as compared to those cases in which he included the pylorus.

Reports of Cases Illustrating Unusual Features

Case 1: A white farmer, aged 40, had had a gastroenterostomy elsewhere two years before admission. Nausea and vomiting started before he left the hospital. Subsequently the attacks became associated with violent abdominal pain, distention and borborygmus. The abdomen showed marked distention above the level of the umbilicus and exaggerated visible peristalsis proceeding from right to left. Gastric analysis showed no free hydrochloric acid. Because of obvious obstruction, there was no attempt made to get x-ray visualization. One year previously the x-ray films had shown a rapidly emptying of the stomach through both the pylorus and gastroenteric stoma, but no ulcer crater.

At operation, Jan. 19, 1926, the pylorus and duodenum were patulous with a small ulcer scar in the latter. The gastroenterostomy revealed an abnormally long afferent loop, tremendously dilated and hypertrophied, involving not only the jejunum but also the duodenum. The stoma was large and well placed. In the efferent jejunal loop about two inches below the stoma there was an ulcer with cicatrization and partial obstruction. The gastroenterostomy was disconnected and a lateral entero-anastomosis between the dilated afferent and the small efferent jejunal loops performed. The marked difference in the respective sizes of the jejunal loops made any other procedure almost impossible. Convalescence was uneventful. He was discharged on the twenty-first day. A few months later he had gained 29 pounds and has remained well to date.

Comment: The cause of the jejunal ulcer in this patient was clearly an error in surgical technic. The surgeon had made a posterior gastroenterostomy with an extraordinarily long afferent loop. This was at first followed by retention in the afferent loop, vomiting and vicious circle. In the course of time a jejunal ulcer with cicatrization and true obstruction just beyond the stoma developed.

Case 2: A woman, aged 57, had had a gastroenterostomy elsewhere in 1923 for duodenal ulcer. Following this there was freedom from symptoms for a period of only two years. Then for an interval of about 14 years, from 1925 to 1939, she suffered ulcer pain with food and soda ease, and with periods of remission. Her case was brought to a sudden climax in March, 1939, by a profuse gastric hemorrhage. After she had recovered from this, x-ray films showed a small ulcer crater in the gastroenteric stoma with the barium passing from the stomach both by the pylorus and stoma. She was discharged from the hospital on an ulcer regime. After five months she was readmitted with a return of ulcer symptoms, which could not be controlled.

At operation, in September, 1939, the pylorus and duodenum were found widely patent with an ulcer scar in the latter. The gastroenterostomy was disconnected and a small, shallow jejunal ulcer excised. The convalescence was uneventful. She was discharged on the sixteenth postoperative day and is now free from symptoms.

COMMENT: The problem in this case was the decision for or against radical gastrectomy after the gastroenterostomy was disconnected. The jejunal ulcer was surprisingly small, hardly enough to justify symptoms which had been present for so many years, in a lesion which is ordinarily so progressive. Therefore, with a small ulcer, probably of recent origin, and low gastric acidity, gastrectomy was omitted. In addition the scar in the duodenum was small and inconspicuous, suggesting that the original gastroenterostomy might have been without good indication. At any rate it was deemed safer under all of these circumstances to await clearer indications before undertaking a radical procedure. At the present time the patient reports that her digestive processes are more comfortable than they have been for many years.

CASE 3: A salesman, aged 49, had been operated upon elsewhere in 1925 for an acute perforation of a stomach ulcer. In 1929 he was operated upon by another surgeon for a second acute perforation. On admission, in 1932, he had a pyloric stenosis with about 50 per cent gastric retention.

At operation, May, 1932, the duodenum was adherent to the under surface of the liver and involved in a large, inflammatory mass with cicatrization and stenosis. A posterior gastroenterostomy was done with some difficulty because of many adhesions involving the posterior stomach wall. The convalescence was uneventful and he was discharged on the seventeenth postoperative day. Two years later, 1934, he returned with ulcer symptoms, after having enjoyed freedom from symptoms for almost two years. The x-ray films showed a rapidly emptying stomach through the gastroenteric stoma and the usual irregularity around the stoma. There was high acidity in the gastric contents. He refused hospitalization, but a short time later was re-admitted with an acute perforation from a marginal ulcer, from which he died after operation, on the ninth day.

COMMENT: The case presents the exception to the general rule that gastroenterostomy in the presence of pyloric stenosis can be expected to give good results with a minimum of fear of gastrojejunal ulcer. It is also worthy of note that he had high gastric acidity when the marginal ulcer occurred. The case also suggests a very extraordinary degree of tissue susceptibility to ulceration—three acute perforations with an ulcer experience covering 22 years.

CASE 4: In 1917, a white nurse, aged 35, had a gastroenterostomy elsewhere for peptic ulcer. In 1931 another operation, the exact type of which could not be learned, was done for a recurrent ulcer. In 1938 she had another gastroenterostomy for ulcer. In September 1939, she was suffering intense and constant pain in the left upper quadrant and was very much emaciated. The x-ray films showed a penetrating ulcer at the gastroenteric stoma.

At operation, Sept. 2, 1939, a large, indurated, perforated ulcer adherent to the anterior abdominal wall was found in the stoma of an anterior gastroenterostomy. The first portion of the duodenum was a dense mass of cicatricial tissue with complete occlusion of the lumen so far as palpation could

determine. Adhesions were widespread. Also it was apparent that a posterior gastroenterostomy had been previously disconnected. There was no entero-anastomosis. The anterior gastroenterostomy was disconnected and a radical, antecolic gastrectomy performed, supplemented by an entero-anastomosis. The distal division of the stomach had to be proximal to the pylorus, since section through, and closure of the sclerotic duodenal stump would have been impossible. With the pylorus open it was just possible to pass a small probe through the lumen of the duodenum. Except for a superficial wound infection, the patient made an uneventful convalescence and was discharged on the twenty-first postoperative day.

Fig. 1. Film shows a chronic perforating ulcer at the stoma of a gastroenterostomy with the base of the ulcer adherent to the anterior abdominal wall. Complete obstruction of the pylorus, site of primary ulcer, is seen. (Case 4.)

COMMENT: It is now generally agreed that of all the operative procedures for marginal ulcer following posterior gastroenterostomy, a procedure which disconnects the posterior gastroenterostomy and supplements an anterior gastroenterostomy is the most unsound, and is followed by a high incidence of recurrent ulcer. This had been the procedure in this case. In extenuation of the procedure, it must be noted that the patient had an extreme degree

of duodenal stenosis. Indeed at the operation considerable anxiety was felt about the pouch of pylorus which had only the almost indistinguishable duodenal channel to drain its secretions. The conclusion that this patient had a high degree of tissue susceptibility seems unquestionable with an ulcer experience of 22 years. While she is well to date with no free acid, the gastrectomy may not protect her from another ulcer.

Fig. 2. Film shows a large ulcer crater at the gastroenteric stoma with marked contraction of the stomach wall opposite the stoma. This ulcer proved to be microscopically malignant. (Case 5.)

CASE 5: A farmer, aged 57, had had a gastroenterostomy in June, 1927 for a chronic perforating duodenal ulcer. Following this he was symptom free for a period of 11 years. Ten months before admission in April, 1939, he had begun to suffer pain in the upper left quadrant of the abdomen with loss of weight and strength. The gastric acidity was low and the x-ray films showed a large ulcer crater at the gastroenteric stoma with considerable contraction of the stomach in the region.

At operation, April, 1939, a large, freely movable, edematous apparently inflammatory, mass with enlargement of the regional lymph nodes was found at the gastroenteric stoma. The gastroenterostomy was disconnected and a

radical gastrectomy of the posterior Polya type was done. When the gastroenteric stoma was exposed to view, an exceedingly large ulceration was situated at the site of the anastomosis, part of the ulcer being in the stomach and part in the jejunum. Convalescence was complicated by an enteric fistula which appeared on the eighth postoperative day, and closed spontaneously by the thirty-fifth day. He was discharged two and a half months after operation. To date he is in good health and free from symptoms.

Fig. 3. Photomicrograph showing adenocarcinomatous change in a chronic gastro-jejunal ulcer. (Case 5.)

COMMENT: The unusual feature in this case is that the pathologic diagnosis of the ulcer was carcinoma, with only inflammatory changes in the lymphatic glands. Carcinomatous degeneration in a jejunal ulcer has previously been reported by Singer[18] and he refers to three others in the literature. Another feature of the case is the long period after the gastroenterostomy with freedom from symptoms before the gastro-jejunal ulcer developed, and the presence of a very low gastric acidity.

CASE 6: A textile worker, aged 52, had had a posterior gastroenterostomy in 1923 for perforating duodenal ulcer. In 1933, ten years later, he was examined and a diagnosis of jejunal ulcer made. He was not seen again until 1939 when he presented the classical symptoms of a gastro-jejuno-colic fistula. The symptoms had been present for about six months. The x-ray films showed the barium enema passing from the transverse colon into the stomach in the region of the gastroenteric stoma.

At operation, Nov. 23, 1939, the pyloric ring and duodenum were wide open with a scar in the first portion of the duodenum. In the region of the gastroenteric stoma the stomach, transverse colon and jejunum were all in close proximity. A large opening sufficient to admit the index finger between the lumen of the transverse colon and the stoma could be easily palpated.

Fig. 4. Gastro-jejuno-colic fistula. The film shows the barium enema having filled the colon, passed through the gastro-jejuno-colic fistula, and partly filled stomach which is the shadow seen just above that of transverse colon. (Case 6.)

The walls of the jejunum and colon showed no edema, redness or other changes. Because of the large size of the fistulous opening and the poor condition of the patient a two stage operation after the plan suggested by Lahey was undertaken. The stomach was divided above the stoma, the open end was anastomosed to the jejunum in front of the colon and the distal end containing the stoma, fistula, etc., closed. Later when the patient had improved, the distal segment was to have been resected with disconnection of the gastroenteric anastomosis and closure of the opening in the colon. The second stage of the operation was never reached. The patient's condition never justified it and he died nearly two months after the primary operation.

COMMENT: Autopsy revealed that massive fecal reflux and accumulation had filled the closed stomach pouch, the duodenum, and

part of the proximal jejunum with a secondary liver infection and death from toxemia. Although the patient lived for a long while following the operation, he never really made any improvement, but grew more emaciated and weak. The incision healed, the bowels moved normally, he took his diet and even got out of bed for a while. He suffered periodic paroxysms of abdominal pain and there were physical signs of accumulation in the closed stomach loop easily made out on the thin abdomen. The extremely large size of the fistulous opening, I think, added the greatest degree of gravity to this case.

SUMMARY

In addition to a brief review of the subject of postoperative gastro-jejunal ulcer, six cases are reported, each presenting a different phase of the problem of causation and treatment. All six cases followed the surgical treatment of duodenal ulcer. In the first two cases, the cause of the jejunal ulcer may be ascribed to operative errors at the primary operation. Neither case was treated by a radical gastrectomy. The third and fourth cases both had gastro-enterostomies in the presence of well marked pyloric stenosis, each developed marginal ulcer, each had four operative experiences for ulcer. With a history covering more than 22 years, a high degree of tissue susceptibility must have been present in each. The fifth case developed a malignant gastro-jejunal ulcer after eleven years of freedom from symptoms following gastroenterostomy. The last case had a marginal ulcer ten years and which finally ruptured into the colon.

623 Professional Bldg.

REFERENCES

1. Balfour, D. C.: Recurring Ulcers Following Partial Gastrectomy, Ann. Surg. *88:* 548-553 (Sept.) 1928.
2. Balfour, D. C.: Results of Gastro-enterostomy for Ulcer of the Duodenum and Stomach, Tr. Am. S. A. *48:* 146-150, 1930.
3. Berg, A. A.: Report of the End-results of Subtotal Gastrectomy for the Radical Cure of Gastric and Duodenal ulcer, Tr. Am. Gastro-enterol A. *26:* 226-232, 1933.
4. Berg, A. A.: Mortality and Late Results of Subtotal Gastrectomy for the Radical Cure of Gastric and Duodenal Ulcer, Ann. Surg. *92:* 340-366 (Sept.) 1930.
5. Edkins, Quoted by Ogilvie, W. H.
6. Eggers, Carl: Gastro-enterostomy, Ann. Surg. *108:* 84-98 (July) 1938.
7. Finsterer, H.: Results of Repeated Operations upon the Stomach Especially for Gastro-jejunal Ulcers, Surg., Gynec. & Obst. *68:* 334-346 (Feb. 15) 1939.
8. Grossman, Abraham: Post-operative Jejunal Ulcer; Study of 23 Cases Operated upon at Presbyterian Hospital, New York, Ann. Surg. *108:* 105-123 (July) 1938.
9. Heuer, G. J.: Choice of Operations in Treatment of Peptic Ulcers, New York State J. Med. *35:* 1-9 (Jan. 1) 1935.
10. Hunt, V. S.: Lateral Gastro-duodenostomy in Certain Cases of Duodenal Ulcer and Recurring Ulcer, Ann. Surg. *110:* 622-628 (Oct.) 1939.

11. Hurst, A. F., and Stewart, M. J.: Jejunal and Gastro-jejunal Ulcers, Lancet *2:* 742-748 (Oct. 13) 1928.
12. Kelly, R. C.: Recurrent Peptic Ulceration, Lancet *1:* 1-5 (Jan. 7) 1939.
13. Lahey, F. H., and Marshall, Samuel: Technique of Subtotal Gastrectomy for Ulcer, Surg., Gynec. & Obst. *69:* 498-507 (Oct.) 1939.
14. Lahey, F. H., and Seinton, N. W.: Gastro-jejunal Ulcer and Gastro-jejuno-colic Fistula, Surg., Gynec. & Obst. *61:* 599-612 (Nov.) 1935.
15. Luff, A. P.: Collective Investigation into the After-history of Gastro-enterostomy, Brit. M. J. *2:* 1074-1078 (Dec. 7) 1929.
16. Ogilvie, W. H.: Place of Surgery in the Treatment of Peptic Ulcer, Lancet *1:* 419-424 (Feb. 23) 1935.
17. Rienhoff, W. F., Jr.: Sympathetic Nerve Block as an Adjunct Anesthesia in Minimal Resection of the Stomach for Peptic Ulcer, Ann. Surg. *110:* 886-901 (Nov.) 1939.
18. Singer, H. A.: Carcinoma of the Gastro-jejunal Stoma Following Operation for Peptic Ulcer, Arch. Int. Med. *49:* 429-438 (March) 1932.
19. Walters, W. W.: Gastro-jejunal Ulcer, a Study of 155 Cases, Am. J. Surg. *46:* 83 (Oct.) 1939.
20. Walton, J.: Quoted by Fogelson, S. J.: Gastro-duodenal Ulcerative Disease, Review of the Literature for the Years 1934 to 1936, inclusive, Internat. Abstr. Surg. *65:* 1-19 (July) 1937.

PROGRESS AND DEVELOPMENT IN THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

FRANK S. JOHNS, M. D.
Johnston-Willis Hospital
Richmond

SURGICAL progress in the treatment of any disease is difficult to record. Especially is this true of pulmonary tuberculosis, a disease of known origin, and considered until recently to be exclusively medical. Here, the varieties of surgical procedures have been numerous, with each advocate claiming so much success for his type of treatment, we are tempted to agree with Josh Billings' memorable comment: "I wouldn't mind people knowing so much if they just didn't know so much that isn't so!"

All error granted, great strides have been made in surgical collapse therapy. The principle is not new. Many refinements in this type of surgery prove real progress, and undoubtedly much is still to be accomplished in its development. Today a close study of the subject, and years of surgical experience in this field, prove definitely that there is no standard operation for the surgical treatment of pulmonary tuberculosis. A given case with a cavity in a certain location must have an operation fitted to the condition of the patient and *to the location of the cavity*. It may even be said authoritatively that the only standardized feature of this surgical treatment is the fact that it must be *selective*, that every operation should be correctly selected for the specific case and his specific cavity or cavities.

Our first and most important point for discussion is the use of pneumothorax. This important procedure is too often delegated to some one of limited experience, when it should be handled by the most capable member of the staff. Within the past decade thoracoplasty and other less extensive operations for the purpose of closing cavities have received liberal consideration from the surgeon. Pneumothorax has been somewhat pushed into the background; but this surgical procedure, and it is truly surgical, should always go in the forefront of our crusade against pulmonary tuberculosis.

The unfortunate practice of prolonged observation of tuberculous patients with cavities has happily passed into the discard. A prompt pneumothorax when indicated, given early in the disease, is the most effectual procedure that can be used in the treatment of pulmonary tuberculosis. It hastens recovery, and by increasing our turnover in sanatoriums, saves untold expense both to the patient and the public. If our state health departments would designate an

Read before the Postgraduate Surgical Assembly (the eleventh annual meeting) of The Southeastern Surgical Congress, Birmingham, March 11, 12 and 13, 1940.

alert, intelligent, trained physician to the simple duty of seeing to it that these patients get the proper treatment promptly, and that they are got out of the hospital when the treatment is complete, it would be a great public economy. I am convinced that our bed capacity would be advanced by one third and our state health bill consequently reduced.

We must not only see to it that our tuberculous patients get their pneumothorax early; it must also be continued until the patient is well. This is of great importance. A too frequent error in allowing the lung to expand before the patient is free from symptoms is often responsible for thoracoplasties and other types of chest surgery.

An astounding number of cases are reported to have had some type of phrenic surgery. It is unfortunate that this minor surgical procedure can be done so easily, and that even when not used properly or advisedly so little apparent damage follows it. The results reported by different authors are sufficient to show that the operation is abused. Graham states phrenicotomy cured 4 per cent of his series; while O'Brien claims that the same procedure closed the cavities in 50 to 55 per cent of his cases. We wonder how two surgeons could be so far apart in their results from the same operation for the same disease. A few years ago, I read a paper before the Virginia Medical Society calling attention to the too liberal use of this operation. I am still firmly of the opinion that there is a distinct place for operations on the phrenic nerve in the treatment of pulmonary tuberculosis. I think that crushing the nerve should be the procedure of choice. But, I am frank to say that I know this operation is done too often, and that its usefulness is limited.

The changes in technic and the achievements accomplished by thoracoplasties make interesting history. The resection of ribs over tuberculous cavities for closure is acknowledged to the ingenuity and courage of DeCerenville. In 1885 he was the first to resect small sections of "two to three" ribs over a diseased area. His plan was sound, and had he carried his idea a little further, he would actually have had what we are doing today under the name "selective collapse." We note that he made no mention of beginning his operation at the base or apex of the thorax. His plan was to collapse the cavity and he naturally advocated resecting the ribs over that particular area.

From there the record is a full one. Pressing on boldly from DeCerenville's cautious experiment, the pendulum had swung off by the early 1920's to the immense, ill-named "standard" operation, preached by Sauerbruch and practiced until fairly recently by his disciples. Thence, within the past ten years, it has swung back.

Today we have the paradox of our radically conservative *selective collapse therapy.*

Fifty-five years ago DeCerenville probably removed only short sections of his "two to three ribs." He was feeling his way, like a good master surgeon, and he doubtless realized as we do that the closure of cavities was the essential thing. Unfortunately for the development of the treatment of pulmonary tuberculosis, this chief surgical objective became for a time obscured. A gigantic "complete" procedure gained great popularity. It was dramatic surgery, involving the resection of large portions of the first to the eleventh ribs, inclusive; and it purported to cure the patient's pathology by the "complete collapse" of his entire lung. Many a tuberculous thorax was conscientiously mutilated; much vital healthy tissue was sacrificed.

When his results were not favorable, the advocate of "complete thoracoplasty" could always fall back on the condition of his patient's opposite lung, his bilateral involvement, lack of fibrosis, etc. But then, as now, the real problem, though temporarily forgotten, was the closure of the cavity or cavities, with a second not less vital factor for consideration, namely: the surgical risk. Will the patient stand the operation?

Clinical experience has taught us that middle-aged patients stand rib resection poorly, that women follow the usual rule of taking any type of surgery better than men. As we should expect, lesions of the right side carry a high mortality and operations done at the base first have the higher death-rate. (Note: I have done no operations at the base since 1926.) The best guide to follow in selecting cases suitable for a major operation such as thoracoplasty is an astute physician, thoroughly schooled in the treatment of tuberculosis. Such a man has followed his cases for years and knows their changing conditions. He will know when the optimum time for operating has arrived, and what has happened in the way of fibrosis and activity.

Today we understand that bilateral lesions are practically always present—though it has been said that the word always should not be used in surgery. Further, we have learned to accept the fact that when tuberculous cases are referred to a surgeon they have been sick for years, not days or months. There are no early operations in tuberculosis. Therefore, when tuberculous cavities come under surgical treatment, we have little reason to worry about whether or not the mediastinum is fixed. Most certainly it is. And this fixed condition is no contraindication to a resection of numerous ribs. This is only one of the many problems that confront us in doing a thoracoplasty.

In 1925 a patient with an apical lesion was referred to me for thoracoplasty by Dr. Dean B. Cole. I explained to her that it would be necessary to do two or more operations on her for a cure. Until then, I had followed the operation as described by European surgeons and practiced in this country by all leading thoracic surgeons. My patient told me in a very impressive manner that she would submit to one operation only.

In consultation with my medical colleague, Dr. Cole, I decided to resect ribs over the cavity which was in the apex first, with the idea of closing it. Under combined local and gas anesthesia, a selective thoracoplasty was done. The result was surprising. The patient's expected stay in the hospital was cut in half; the cavity was closed, and the patient soon returned to earning her own livelihood. She is well today, fifteen years later. Since that time, I have done selective collapse on all my cases, selecting the operation to suit the case and the location of the disease.

In selective collapse we no longer ask whether a thoracoplasty should begin at the base or at the apex. It should begin in the most suitable location to close the cavity with the least possible harm to the healthy lung tissue. Certainly as much attention should be given to preserving the good part of a lung as to ridding the remaining portion of its disease.

In 1929, with Dr. Cole, I presented a paper on these findings before the Association for Thoracic Surgery in St. Louis. It was entitled "The Results in Selective Thoracoplasty" and advanced our grounds for advocating this type of collapse therapy for pulmonary tuberculosis. It was received in frankly dubious silence. Two years later, a further report was made to the same scientific body at Ann Arbor. Its reception there was scarcely more enthusiastic. But in 1940 it is gratifying to me to read that my recommendation has been adopted in toto by American thoracic surgeons.

Every surgeon is familiar with stages of operations. It means simply a device for doing the necessary operation with safety to the patient. And so it is with extrapleural thoracoplasty. We must simply consider the condition of the patient, and perform it in as few stages as will be safe for him.

It is a fact too well known that no matter how completely we think a thoracoplasty may have been carried out, a certain per cent will have to undergo another operation to close remaining cavities. Overholt states that he has had to do some type of revised operation in 20 per cent of his cases. I believe that his percentage is unusually low. To close cavities which have never really been closed, is the bugbear of thoracoplasty. It is the most difficult task in thoracic surgery. Many writers have suggested how it should be done, but

no method has proved satisfactory. I feel that the time to arrange for closing secondary cavities is at the primary operation. Overholt has combined the good points from Holst's and Semb's procedures for freeing the upper lobe and allowing the apex to descend, thereby offering a better opportunity for adequate closure of cavities. In this manner, the compression will be made from above as well as laterally, and there will be fewer tongue-like cavities left back against the spine.

For many years, Dr. Cole has used lipiodol to demonstrate questionable cavities. When in doubt, this is a good method for bringing out remaining defects, and it will explain many unexplained positive sputums. Improvement in x-ray makes it possible today to photograph the chest in planes, thus revealing further cavities otherwise overlooked.

For a long time there has been a need for some type of collapse therapy in the apical region of cases not affected by pneumothorax—and too ill for selective thoracoplasty. Apicolysis with a variety of foreign substances was not entirely satisfactory. It has been replaced, certainly to some extent, by extrapleural pneumothorax. This is a simpler and a less troublesome operation, in properly selected cases, although unquestionably this procedure has a limited field.

The use of bronchoscopy in thoracic surgery is becoming more liberal each year. The demand for its use and the technical skill with which it is used has made the procedure essential to a well-conducted thoracic organization. Its diagnostic and therapeutic value is indispensable to both the medical and surgical staffs.

Our mortality in the surgery of pulmonary tuberculosis will be influenced by the selection of cases, the preparation, and the extent of the operation. When the internist and surgeon have agreed that a patient is suitable for surgical collapse, the preparation of the patient should receive every consideration. If the patient presents a grave risk, a transfusion should be given prior to the operation and another one given during the operation. The extent of the operation should not be decided beforehand, but should be determined by how the patient reacts to his operation. The surgeon should always stop the operation if there is any question about the risk. Operations on the thorax may be stopped at any stage. There is no sound reason why this fact should not always be kept before us. I might mention the question as to how much time should elapse between operative stages. Unfortunately, an attempt has been made to state what this interval should be. Experience has taught me that any attempt to fix a stated time is very unfortunate, and costly to the life of the patient. After he has come through the first stage of his operation,

there is no reason why a liberal opportunity should not be given him to recover completely. He should be in just as good condition as he was at the previous operation. Judgment here, in deciding when to proceed with further surgery, has a distinct influence in the outcome of the case.

In my operations on the chest, I used only local anesthesia. At the present time, I use local and inhalation anesthesia with a limited amount of ether: the results are equally good. I think, however, the way it is administered is quite as important as the type of anesthesia used. An expert anesthetist is essential for few respiratory complications.

The Southern Surgeon

Published Monthly by
The SOUTHERN SURGEON PUBLISHING COMPANY
701 Hurt Building
ATLANTA

L. MINOR BLACKFORD, M.D.
Editor

B. T. BEASLEY, M.D.
Managing Editor

WALTER G. STUCK, M.D.
Associate Editor

Subscription in the United States, $5.00

VOLUME IX AUGUST, 1940 NUMBER 8

MEDICAL PREPAREDNESS

Each member of the two organizations behind THE SOUTHERN SURGEON has recently received a questionnaire from the Committee on Medical Preparedness of the American Medical Association. The Chairman of this Committee, it is interesting to note, is also President of The Southeastern Surgical Congress: we are sure that Dr. Abell is the man for the new job.

It is not fitting to try here to stimulate the patriotism of the medical profession, nor to boast of it. The patriotism of the sons and grandsons of Confederate soldiers is certainly not second to that of any other Americans. But doctors are busy men and they have been known to put aside questionnaires to be filled out in favor of matters demanding their immediate attention. If any surgeon in the South has not yet returned his completed form to Dr. Abell's Committee he must recall that in 1940 time is of the essence: we must let the world know that America will act as one man if need arise.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

THE FUNDAMENTALS OF INTERNAL MEDICINE. By WALLACE MASON YATER, A. B., M. D., M. S. (in Med.), F. A. C. P., Professor of Medicine and Director of the Department of Medicine, Georgetown University School of Medicine; Physician-in-Chief, Georgetown University Hospital; Physician-in-Chief, Gallinger Municipal Hospital, Washington, D. C.; formerly Fellow in Medicine, The Mayo Foundation. First Edition, Revised. 1,021 pages, with 255 illustrations. Price, $9. New York and London: D. Appleton-Century Company, Inc., 1940.

This book has been termed by an experienced teacher, "the first modern textbook of medicine." There is a tendency to dogmatism, as is necessary when so much is covered in such short space, and therefore one is tempted to take issue with the author as to a detail here and there. However the point of view from which the book is written, with the emphasis on physiology, biochemistry and pathology, and the superb condensation of material enable us to understand why it has been so rapidly re-issued, and make us wish to add our meed of praise.

"The Fundamentals of Internal Medicine" is especially recommended to the following groups:

1. Undergraduates in the clinical years who may wonder what their studies have to do with the practice of medicine, as well as to those who are beginning the study of clinical medicine and who are in danger of being unable to see the forests for the trees.

2. Those preparing for the examinations of the specialty boards.

3. Young doctors who need to keep their clinical work in touch with the hard rock of scientific practice.

4. Surgeons who need the latest dope on medical aspects of their cases.

5. All of those practitioners who are in need of a refresher course; me and just about all the other doctors I know who haven't given up their ambition to practice good medicine.

THE FOOT AND ANKLE: THEIR INJURIES, DISEASES, DEFORMITIES AND DISABILITIES. By PHILIP LEWIN, M. D., F. A. C. S., Associate Professor of Bone and Joint Surgery, Northwestern University Medical School; Professor of Orthopedic Surgery, Post-Graduate Medical School of Cook County Hospital; Attending Orthopedic Surgeon, Cook County Hospital; Attending Orthopedic Surgeon, Michael Reese Hospital; Consulting Orthopedic Surgeon, Municipal Contagious Hospital, Chicago, Illinois. 620 pages, with 303 illustrations. Price, $9. Philadelphia: Lea & Febiger, 1940.

Twenty-five years ago, the author tells us, Dr. Kanavel suggested to him that he write a book on the foot along the lines of Kanavel's work on the hand; that is, "a book that will tell the general practitioner what to do for the common conditions that occur in and around the foot." It is to be regretted that Dr. Kanavel did not live to see this fruition of his suggestion. Another surgeon who has greatly influenced Dr. Lewin and who we are glad

to note is still alive is Dr. Michael Hoke. Much of Dr. Hoke's teaching is incorporated together with many of his illustrations, many not previously published. It is nice to have that part of his important pioneer work dealing with the foot assembled in one book.

Although designed primarily for the general practitioner, the reviewer cannot but believe that this well rounded, thoroughly illustrated, book will prove valuable to the orthopedic surgeon, if only as a ready reference. He would like to comment especially on the chapter dealing with neurologic lesions affecting the foot and ankle and to quote a sentence, "The only equipment required to test for neurological conditions is a pin, percussion hammer, a wisp of cotton, hot and cold water in test tubes, a sense of touch and a moderate amount of intelligence." That sentence typifies both the common sense and the gift of expression that appear on every page.

CLINICAL HEART DISEASE. By SAMUEL A. LEVINE, M. D., F. A. C. P., Assistant Professor of Medicine, Harvard Medical School; Senior Associate in Medicine, Peter Bent Brigham Hospital, Boston; Consultant Cardiologist, Newton Hospital; Physician, New England Baptist Hospital, Boston. 495 pages, with 109 illustrations. Second Edition, Revised and Reset. Price, $6. Philadelphia and London: W. B. Saunders Company, 1940.

Writing simply, clearly, conversationally, dogmatically, Dr. Levine has provided the practicing physician with a book in which he can rapidly learn exactly what the author thinks about the various aspects of heart disease. And what he thinks in this line is authoritative: the most captious critic will have to look hard to find even immaterial minutiae to quibble about.

There are no illustrations except electrocardiograms but these are carefully chosen and clearly interpreted. The author emphasizes that, though the electrocardiogram is of the greatest value in some cases, it has many limitations and that one must not attach great significance to minor variations from the classic configurations.

TEXTBOOK OF GENERAL SURGERY. By WARREN H. COLE, M. D., F. A. C. S., Professor of Surgery, University of Illinois College of Medicine; formerly Associate Professor of Surgery, Washington University School of Medicine, St. Louis, and ROBERT ELMAN, M. D., Associate Professor of Surgery, Washington University School of Medicine, St. Louis. Second Edition. 1,031 pages with 559 illustrations. Price, $8. New York and London: D. Appleton-Century Company, 1939.

This volume is a direct outgrowth of lectures in surgery given by the authors to third and fourth year medical students at the Washington University School of Medicine. Doctors Cole and Elman appreciate that a good surgeon must have a thorough knowledge not only of gross but also of microscopic pathology. For this reason they have wisely seen fit to attach photomicrographs of various neoplastic and other diseases to the illustrations of the gross lesions.

The authors point out that the age-old use of an ice cap in the conservative treatment of appendicitis is based on a misconception and may at times be harmful.

The chapters on the Alimentary Tract, Liver, Gallbladder and Spleen, Peritoneal Cavity, and Fractures, Dislocations and Sprains are especially

good. The material of the entire book is authoritative and at the same time is presented in such an interesting way that even a sleepy medical student will have trouble laying the book aside.

The illustrations are simple, clear-cut and straightforward with their message.

This book was designed for the use of medical students and general practitioners. Its aim is to impart a thorough groundwork in the more important details of general surgery. Descriptions of operative technic are properly left to systems of surgery. This reviewer's opinion is that Cole and Elman's second edition will be even more popular than was the first. It takes its place among the best of the texts of general surgery.

A Doctor's Holiday in Iran. By Rosalie Slaughter Morton, M. D. Foreword by Surgeon-General Hugh S. Cumming (Retired). 335 pages, with 20 illustrations. Price, $3. New York and London: Funk and Wagnalls Company, 1940.

The American public has ever been partial to success stories. It should prove enthusiastic about this fine story of how Riza, a son of a poor family without formal education or political experience, has so successfully attempted to pull his country through a thousand years of progress in a lifetime.

Dr. Morton, a daughter of old Virginia, has written this account as only a doctor could. Her book should entertain physicians who have not been able to travel so extensively, enable them to forget for a time both the petty worries of their daily work and the greater worries of these troubled times.

THE TEXAS SURGICAL SOCIETY

October Meeting

The regular semi-annual meeting of the Texas Surgical Society will be held in Houston October 7 and 8. The guest speakers will be Dr. A. W. Adson, of the Mayo Clinic, and Dr. Maxwell Harbin, Professor of Orthopedic Surgery of Western Reserve University.